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STUDIES OF GALL-BLADDER FUNCTION

III. A STUDY OF THE ALLEGED IMPEDIMENT IN THE CYSTIC DUCT TO THE PASSAGE OF FLUIDS[1]

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IN 1723, Laurentius Heister described certain anatomical structures in the cystic duct of the human which had not previously been reported. He stated that "in the cystick duct of each . . . he discovered certain beautiful and surprising valves of a spiral figure . . . some in a transverse, some in an oblique situation, which divided the duct, as it were, into a number of cells. These, however, did not entirely close the duct in any part, but were disposed much as the valvulæ conniventes in the jejunum and colon."

Heister states that Glisson and Bianchus believed that these structures "had been framed from imagination," since they were never able to find a real valve in the cystic duct. On the other hand, Bauhine, Bidloo, and Vestus had found them subsequent to Heister's description. He further says: "Schelhammer has gone so far toward it as to allow that the cystick duct will not admit of a style either from the part next the duodenum, or from that next the cystis; but that it very readily admits of inflation either way." And finally Heister, perturbed because the presence of such valves should be questioned after his careful dissections, stated that he "is not afraid to appeal to the publick, whether they are or not valves. Their use also is a subject very worthy to be inquired into." Figure 1 is an illustration of Heister's concept of these structures.

With the advent of renewed interest in the biliary system incident to improved surgical technique, contributions to the physiology of the gall-bladder and new methods of study, the attention of the surgeon, the anatomist, and the physiologist has again been directed to the gall-bladder function. The subject must be studied from numerous angles, and one of these must be a study of the cystic duct, which includes, especially in the human, the heisterian valves. A study of their rôle is a necessary adjunct to a proper concept of gall-bladder function.

Sufficient evidence has accumulated that one may say with little reservation that the gall bladder does, under certain conditions, empty its contents. There are those (1, 2, 4, 15) who believe that the contents are removed without passing back through the cystic duct. There are those who believe that, although the absorption of certain constituents does take place through the gall-bladder lymphatics or blood vessels, the concentrated bile leaves the gall bladder through the cystic duct to enter the duodenum as the result of certain physiological stimuli.

Strips of the gall-bladder wall have been shown to contract (8, 14). The isolated gall

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Fig. 1. "The valves of the cystick duct." (From Heister.)

bladder has been shown to respond to drugs which stimulate smooth muscle contraction (14.) McMaster and Elman (11), working with live dogs, unanæsthetized, and fully recovered from operative effects, report definite spurts of bile from a tube connected to a cannula in the cystic duct, following a meal. They obtained cystic pressures as high as 260 millimeters of bile. In the 5 years elapsing since the wide use of sodium tetraiodophenolphthalein, further evidence has been brought forward to demonstrate the fact that bile must, under certain conditions, leave the gall bladder by way of the cystic duct (7). It is unnecessary to review the entire literature on this subject, but sufficient evidence has been published to warrant the assumption that gall-bladder evacuation through the cystic duct can and does occur.

For the bile from the gall bladder to reach the duodenum by the ductal route, it must pass two possible obstructions, the cystic duct, with its valves of Heister and its cystic kink, and the sphincter of Gage and Oddi.

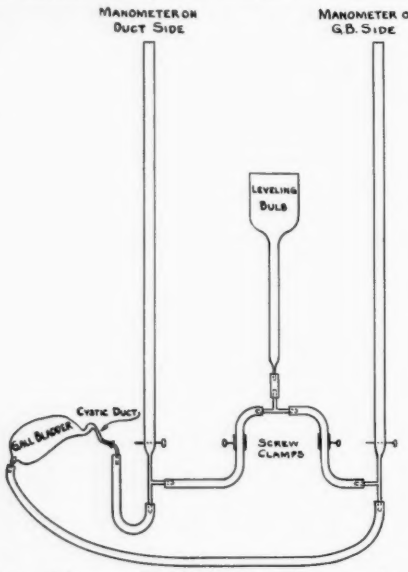

Fig. 2. Diagram of method used in these experiments.

We will not consider the obstruction offered by the sphincter of Gage and Oddi. This subject has been thoroughly studied by McMaster and Elman (12), and their findings are substantiated by work done in this laboratory. The experiments in this report were carried out in order to determine the pressure necessary to force fluid in either direction through the cystic duct in the human cadaver and in the living dog.

Some work of this nature has been done previously. Loehner compared the pressure necessary to cause fluid to flow into the duodenum from the hepatic duct and from the gall bladder. When the sphincter of Gage and Oddi was intact, fluid flowed into the duodenum at the same rate from the gall bladder as from the hepatic duct. With a tube through this sphincter, the flow of fluid from the gall bladder was slower than that from the hepatic duct. Mann stated that the resistance offered to the flow of fluid through the cystic duct does not exceed 30 millimeters of water, but presented no data to substantiate this statement. Mentzer, working with cadavers, concluded that solutions of glucose passed through the cystic duct in either direction with equal facility at pressures of 300 millimeters. However, Mentzer used a pressure higher than that normally attained in the biliary system.

METHODS

Two types of apparatus were used in these experiments. The first consisted of two straight manometers of 4 millimeter bore, a leveling bulb, and cannulas of from 1 to 3 millimeters bore. One cannula connected to a water manometer was tied into the fundus of the gall bladder, and another, also connected to a manometer, was tied into the common

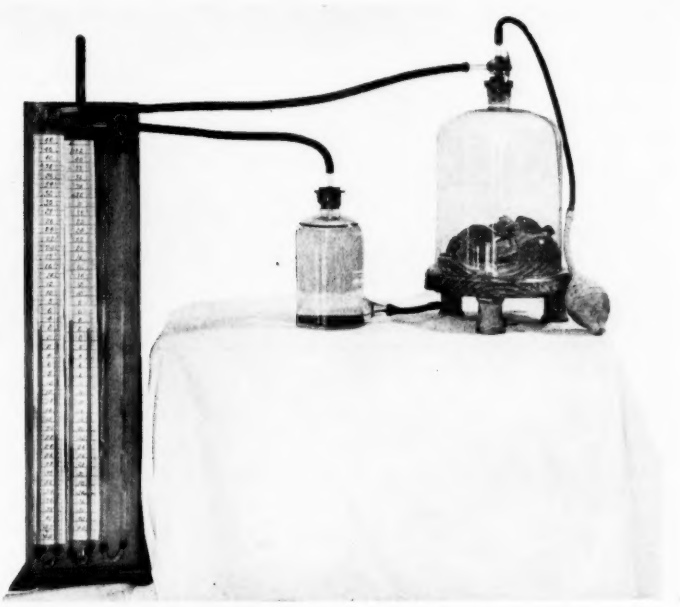

Fig. 3. Bell jar apparatus for altering cholecystic pressures.

duct. The manometers were connected by side arms. A T-tube to which a leveling bulb was attached was introduced between the manometers. By opening a screw clamp between the T-tube and either manometer, the pressure could be changed on one side or the other at will, and the pressure developing on the other side could be determined. The manometers connected in this way are analogous to a simple U-tube, the arms being represented by the two manometers, their connecting base by the gall bladder, cystic, and common ducts (Fig. 2). The zero points of the two manometers were set at the level of the cystic duct. With this apparatus a rise of pressure on one side of the circuit will cause a rise of pressure in the other side if the fluid is free to move through the cystic duct.

In these experiments the pressures were varied on the common duct side of the cystic duct as well as on the gall-bladder side, and the resulting pressures read after about a minute had elapsed. In this way any resistance to the flow of fluid into, or out of the gall bladder could be determined. Pressure changes within the gall bladder were also brought about by causing contraction or relaxation of the gall-bladder musculature by the intravenous injection of pilocarpine hydrochloride or atropine sulphate.

In order perhaps more closely to approximate physiological conditions concerned with the pressure necessary to cause bile flow through the cystic duct, the following method was devised: The apparatus consisted of a bell jar fitted with an air tight junction to a base (Fig. 3). Livers from dogs and from humans at autopsy, with intact hepatic, common and cystic ducts and gall bladder, were used. The specimen was placed within the bell jar. A cannula, connected with a water manometer was tied in the common duct near its entrance into the duodenum, and another manometer was connected to the inside of the bell jar. With this arrangement we had another method of testing the patency of the cystic duct in either direction. It differed from the first method in that the pressure was applied evenly to the outside of the gall bladder instead of on the inside, and

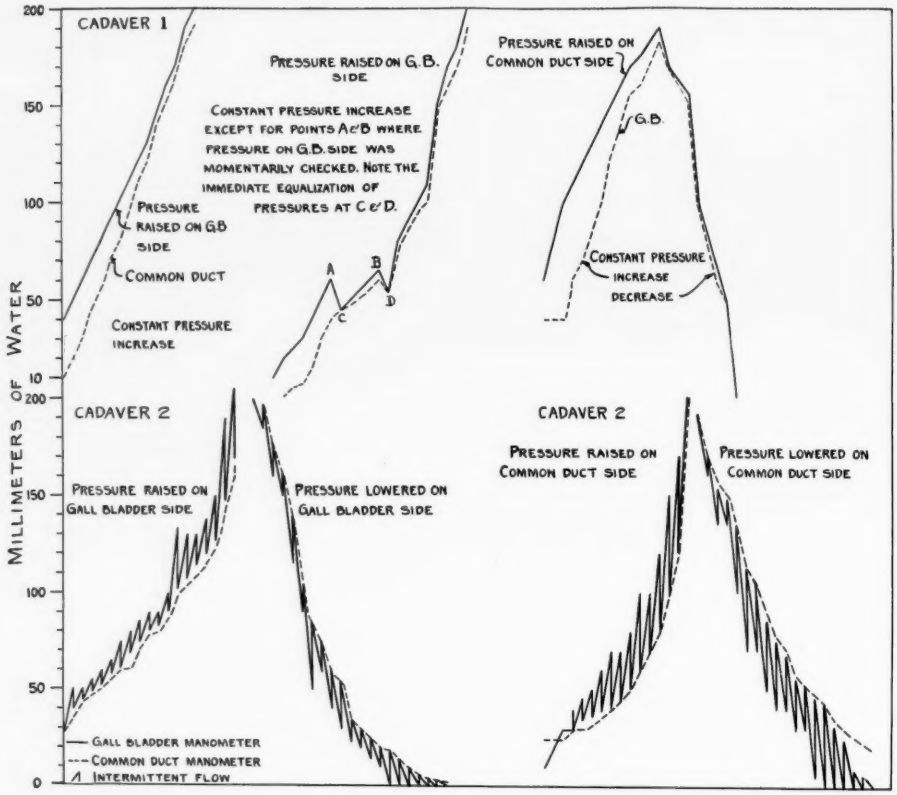

Fig. 4. Cadaver experiments: Upper curves were obtained during the continuous flow of fluid on the gall-bladder or common duct side. Lower curves were obtained during the intermittent flow of fluid.

the fluid which passed through the cystic duct was bile instead of water. One difficulty arises in this type of experiment, in that if pressure is applied to the ducts as well as the gall bladder the ducts, being smaller and containing less fluid than the gall bladder, will collapse. This can be prevented by protecting the ducts with paraffin.

RESULTS

The data were obtained from three well preserved cadavers, five fresh human biliary tract specimens including the liver and duodenum, and from eight dogs which were anæsthetized with sodium amytal, 50 milligrams per kilogram of body weight. In one dog the studies were carried out with the abdomen closed and most of the air removed from the abdominal cavity by the introduction of physiological sodium chloride which was aspirated after closure of the abdominal wound. In the preserved cadavers the dried bile was washed out of the gall bladder, common and cystic ducts by a gentle flow of water. Water was used throughout the system in the case of the cadavers. In the dogs, the bile was not washed out of the system. However, it did not rise into the manometers more than a few centimeters, so that we feel justified in recording our pressures in terms of water pressure.

In the cadavers, a change in pressure in one manometer was nearly instantly followed by an equal change in pressure in the other

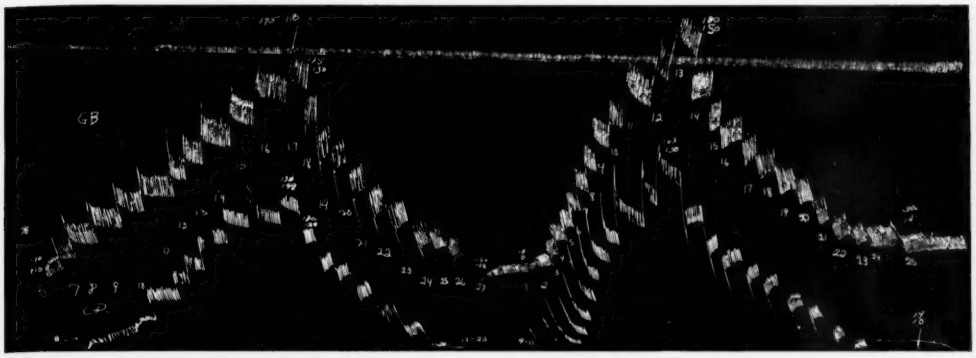

Fig. 5. Tracings obtained with Brodie bellows in the dog. The pressure was increased or decreased intermittently.

manometer, except at pressures around 0 millimeter. In two of the cadavers, the fluid was allowed to flow into or out of one side slowly and continuously, and the pressures in the two manometers read from time to time. The data obtained from the cadavers are shown graphically in Figure 4. It will be noted that the pressure levels tended to equalize even at low pressures. This was true whether the pressure was varied on the gall-bladder or common duct side.

In the dogs, kymograph tracings of the pressure changes were taken, in addition to direct readings. A Brodie bellows attached to the upper end of the manometers gave good tracings with a minimum of dampening effect. Figure 5 illustrates such a tracing. In this animal the results are similar to those obtained from cadavers, the pressure in one side following very closely the pressure in the other.

In the experiments on dogs, when fluid was introduced into the gall bladder through the cannula in its fundus, the pressure rose on the common duct side. It is interesting to note that the greatest pressure difference between the two sides was found when fluid was introduced on the common duct side; when fluid was entering the gall bladder. This might be explained by a gradual filling of the distensible gall bladder.

In three animals, the pressure bulb was placed on the common duct side and the pressure raised and lowered. At lower pressure levels, 0 to 80 millimeters, the response on the gall bladder side of the system to the introduction of fluid on the common duct side varied. Between these points, however, an optimum pressure for filling the gall bladder was reached; and once the optimum was reached, the system acted as a U-tube manometer (Figs. 6 and 7).

When the pressure was lowered on the gall-bladder side to zero, there was never a coincident fall in the pressure on the common duct side to zero. In two cases the pressure fell to 10 millimeters of water. In others the pressure fell to levels varying from 10 to 80 millimeters. Thus, in decompressing from

Fig. 6. Pressure changes in the common duct as a result of changing the intracystic pressure. Dog.

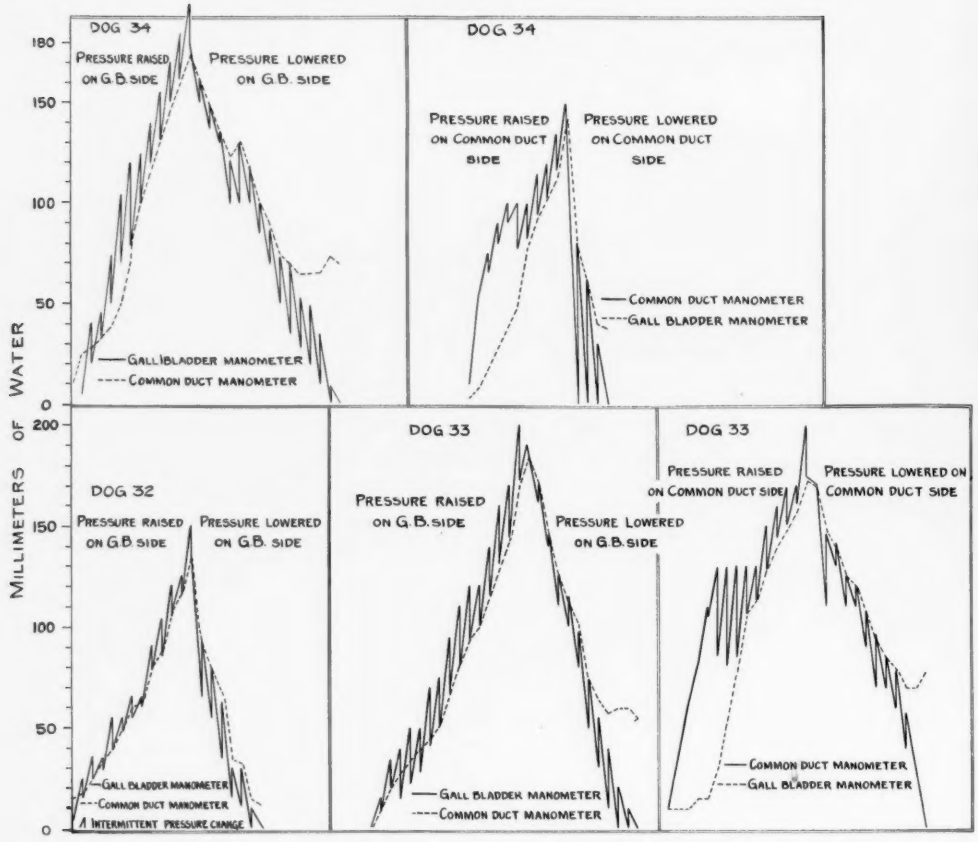

Fig. 7. Pressure changes in the gall bladder and common duct of the dog. The pressures were increased or decreased intermittently.

either the gall-bladder or common duct side, there occurs a lag in the pressure on the side opposite to that from which the fluid is being removed when the pressure on that side drops to 80 millimeters or below. The exact point at which this lag begins varies. This may be due to the effect of a suction action collapsing the ducts.

Likewise, reducing the pressure on the common duct side did not result in so great a decrease in the pressure within the gall bladder, which again may be due to collapse of the small ducts. In none of the five animals did the gall-bladder pressure fall below 40 millimeters, even with pressures on the common duct side below zero values.

When gall-bladder contraction was stimulated with pilocarpine, the pressure rose equally in both the gall-bladder and common duct manometers, and when the muscle was relaxed with atropine, there was an equal fall on both sides (Fig. 8). Likewise forceful pressure on the lower ribs of the dog caused a simultaneous rise of pressure in both systems (Fig. 9).

In the experiments in which the bell jar was used, the pressures on either side of the cystic duct tended to equalize (Fig. 10). Five fresh human gall bladders with their contained bile, were used, and in these there was little or no difference in the pressures in either side of the cystic duct. In all instances, the pressure in

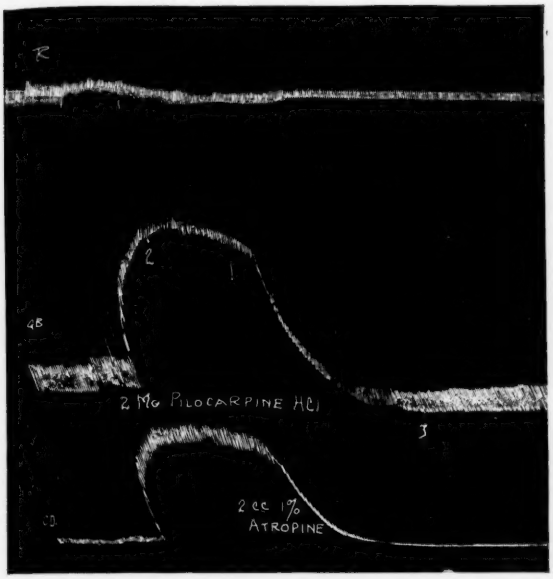

Fig. 8. Intracystic and common duct pressures after the administration of pilocarpine and atropine to the living dog. The gall-bladder pressure reached 210 millimeters of water, the common duct pressure 190 millimeters.

the manometer on the common duct side reaches the level of that on the gall-bladder side after not more than a minute of time. A similar condition was found, however, when a rubber balloon was attached to a twisted glass tube of about 1 millimeter bore; and placed in the bell jar. On account of a similar behavior between this simple artificial system and the gall bladder and its ducts we are led to conclude that the lag found in the equalization of the pressures in our experiments was due to the slow passage of the fluid through the narrow duct system.

DEDUCTIONS

McMaster and Elman (12) found that it required a pressure of 100 millimeters of bile, or more, to cause bile to flow into the duodenum from the common duct. It is evident from our data that in all of our experiments a pressure change on one side of the cystic duct was followed by an equalization of pressures on the two sides when the pressure levels were within the normal range described by McMaster and Elman.

Fig. 9. The effect on gall-bladder and common duct pressures of bilateral compression of the lower ribs.

The variations in our results at lower pressures may well be explained by variations in the size of the ducts through which the fluid had to pass or by collapse of the ducts when pressures were being reduced.

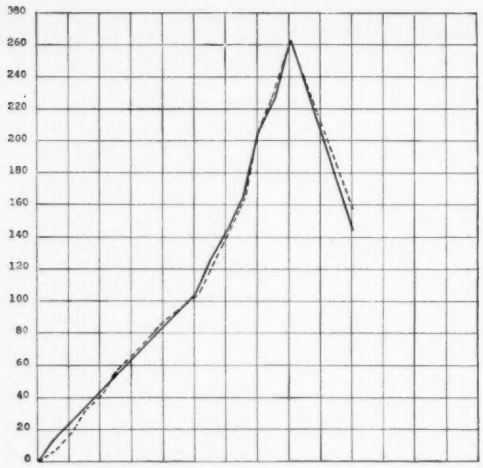

Fig. 10. Bell jar experiment with fresh human material. Pressure readings from common duct following changes in pressure on gall bladder. Straight line indicates pressure in jar; interrupted line pressure in common duct manometer.

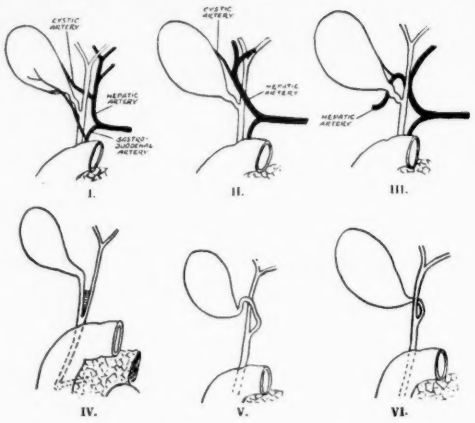

Fig. 11. The variation in the anatomy of the cystic duct. (From Eisendrath). *I*, Cystic artery arises from common hepatic. Accessory cystic artery from gastroduodenal, passing anterior to common bile duct. *II*, Common hepatic artery crosses anterior to hepatic duct and comes to lie in close relationship to the cystic duct. *III*, Right hepatic artery forms a loop behind the neck of the gall bladder. *IV*, Common duct is short. Cystic duct is long and runs parallel with common duct, being adherent to it from some distance. *V*, Cystic duct is long and lies in a loop anterior to the common duct. *VI*, Cystic duct forms a loop behind the common duct.

The anatomy of the cystic duct is quite variable as shown in Figure 11. The curvature in the cystic duct varies considerably; in some instances the duct is almost straight, in others it is kinked; in some it is long, in others short. One of our cadavers had a very kinked duct; in another the duct was short and only moderately kinked. The cystic ducts in the dogs used in these experiments were also quite variable, but no relationship could be found between curvature of the duct and pressure differences on the two sides of the cystic duct.

Objection may be raised to the use of the preserved cadaver and fresh autopsy material for this type of experiment, because the valves of Heister might not offer the same resistance in dead as in living tissues. That this is a possibility is apparent, but it is not possible to approximate more nearly the normal in studying the human cystic duct.

The assertions of Sweet and Jacobson and Gydeson that the kink of the cystic duct offers a direct impediment to the flow of bile might be correct when one estimates this at pressures lower than 80 millimeters of water. It is curious that our experiment which suggests that such is a possibility was carried out on a living dog whose cystic kink was hardly noticeable. However, at pressures above this, we have been unable to determine, either in the human cadaver or living dog, an impediment to the flow of fluid either into or out of the gall bladder.

It should be stated that, although certain observers, in attempting to study this subject, have presupposed the presence of valve-like structures in the cystic duct of the dog, we have not been able to substantiate this. Rudimentary folds are occasionally seen, but we have never observed a structure similar to that seen in the human.

SUMMARY AND CONCLUSIONS

From experiments on seven dogs, we can find no impediment to the passage of fluid through the cystic duct at pressures usually found in the biliary tract. At lower pressures there is, in some animals, an impediment to the passage of fluid from the gall bladder through the cystic duct, which is overcome at pressures of from 10 to 80 millimeters of water.

In three well preserved human cadavers we have found no resistance offered to the flow of

fluid through the cystic duct, nor were we able to demonstrate any in five fresh autopsy specimens.

Two methods for studying this alleged impediment of the cystic duct are described.

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THE HISTOLOGY OF A CASE OF OVARIAN PREGNANCY AT THE END OF THE SECOND MONTH

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OVARIAN pregnancy has been recognized as a theoretic possibility since the beginning of the seventeenth century, when it was first suggested by Mercer (1614). It was nearly three centuries later that the first histological description of an actual case was presented, by Catherine van Tussenbroek (1899). The most complete histological study of an ovarian pregnancy was published in 1908 by Bryce, Teacher, and Kerr. This is also the youngest case yet described, one of about 18 days, and is further unique in that it occurred simultaneously with a normal intra-uterine pregnancy. In their monograph these authors review also 6 other authenticated cases of ovarian gestation.

Ovarian pregnancies constitute a form of abdominal gestation. They may be intrafollicular, or extrafollicular (superficial). The latter differ from ordinary abdominal pregnancies only in that implantation has occurred on the ovary. Intrafollicular pregnancies theoretically may be primary or secondary. In the latter case the fertilized extruded ovum subsequently secures lodgment in a ruptured graafian follicle. The specific character of primary intrafollicular ovarian pregnancy inheres in the assumption that in such cases the ovum is not extruded at the time of rupture of the graafian follicle, but remains within the follicle where it is fertilized, and in the wall of which it becomes implanted. Ovarian implantation involves the excavation of many blood vessels and the consequent production of extensive internal hæmorrhage, accounting thus for the invariable association of a hæmatoma. The case of Bryce, Teacher, and Kerr was one of extrafollicular pregnancy. Implantation occurred adjacent to a large corpus luteum. Tussenbroek's case was an intrafollicular gestation. The case to be described is clearly an intrafollicular pregnancy; whether primary or secondary can not be determined.

Ovarian pregnancies are relatively rare. Strezoff (1927) states that a total of 92 cases have been reported. His own material of 350 cases of ectopic pregnancies operated on since 1912 included only 1 of ovarian gestation, a superficial form. Zimmermann (1927) observed 2 cases among 120 extrauterine pregnancies; 1 intrafollicular, 1 superficial.

The explanation of ovarian pregnancy in terms of the implantation of a fertilized ovum must be correct in the great majority of cases, probably in all cases. The wide occurrence of ectopic pregnancies admits of no other inter-

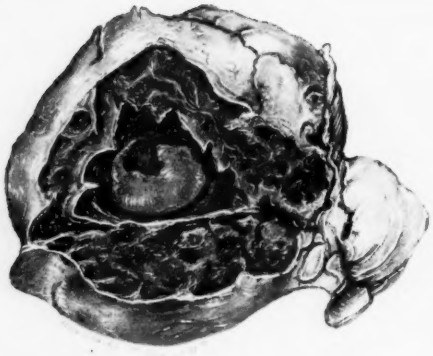

Fig. 1. Drawing of posterior view of left ovary, cut open to reveal the gestation sac with the fetus intact. The stump at the right is portion of the broad ligament. Natural size.

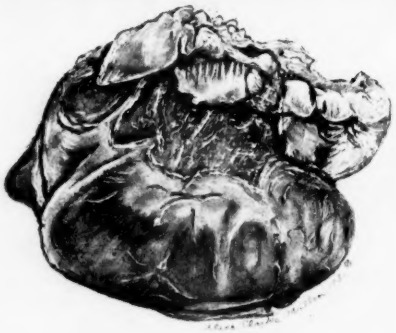

Fig. 2. The pregnant ovary seen from above, attached to the broad ligament. The nipple-like structure at the left is one of the several nodular elevations on the lateral aspect, shown in Figure 3. The nodules represent peripheral portions of the impregnated graafian follicle and contain chorionic villi. Natural size.

pretation. However, the possibility of the parthenogenetic development of an ovum within an unruptured or inadequately ruptured graafian follicle need not be ignored. In view of Leo Loeb's (1911, 1915, 1923) demonstration of segmenting unfertilized eggs in the ovary of the guinea pig, parthenogenetic ovarian pregnancy in the human female may be regarded as at least within the sphere of respectable scientific speculation, as concerns both ovarian gestation and ovarian chorioepitheliomata. Loeb reports parthenogenetic development of ova within the ovary of the guinea pig in approximately 10 per cent of all individuals below the age of 6 months.

Our specimen was secured at operation in the Shreveport Clinic. The operation was done by Dr. B. C. Garrett. Dr. W. J. Norfleet sent the material, a left ovary, to the Laboratory of Histology and Embryology, University of Virginia for microscopical study.

The clinical history led Drs. Garrett and Norfleet to a diagnosis of ectopic pregnancy, confirmed at operation on January 19, 1931. Symptoms first appeared, in the form of severe abdominal pain, on December 11, the approximate date of an expected menstruation. A delayed menstruation began on December 21. Assuming that fertilization occurred at the usual time, about the middle of the menstrual cycle, this ovarian gestation dates from near the day of November 27. The age of the embryo at the time of operation was accordingly, approximately 53 days. However, the histological condition of the embryo indicates that it had been dead for several days. The age at death was close to 50 days. Estimated on the basis of length, namely 15 millimeters, an age of approximately 50 days is again derived. An age of 50 days for this embryo must therefore be correct within a possible error of only several days.

Dr. Garrett reports normal abdominal viscera at operation, "except for the left ovary which was enlarged and hæmorrhagic. The left tube was intact and not adherent to the mass. The mass was easily removed." There was apparently little or no abdominal hæmorrhage.

On median longitudinal section, along the surface opposite the hilum, about a teaspoonful of a clear fluid escaped. The specimen was preserved in a 10 per cent formalin solution.

The specimen, macroscopically apparently an ovarian hæmatoma, has a dark brown color. It has a stout ellipsoidal shape, 5 centimeters in length, 3 centimeters in width, and 3.25 centimeters in thickness (Figs. 1 and 2). The surface contour is uneven, due to a number of irregular domed elevations. Four of these on the lateral aspect of the ovary are especially prominent (Fig. 3). These measure from 5 to 8 millimeters at the base, and have a height at the apex of approximately 2 millimeters. On microscopical examination these nodules were found to consist of a thin wall of ovarian tissue and a core of blood clot with imbedded well preserved chorionic villi (Fig. 4).

The cavity of the hæmatoma has an irregularly oval shape, about 2.5 by 1.0 centimeter. It is lined with a thin chorion, generally atrophic and in certain areas necrotic. In the depth of the cyst appeared an apparently well preserved embryo, 15 millimeters in length (vertex-breech). It was adherent to the floor over the region of the umbilicus. The amnion had ruptured. No trace of the umbilical

Fig. 3. View of lateral border of ovary, showing nodular surface and broad ligament above. Natural size.

vesicle (yolk sac) could be found. The head was clearly indicated by conspicuous branchial arches. Macroscopically the embyro appeared approximately normal. However, the microscopical preparations revealed moderate morphological malformation and profound histological and cytological changes involving widespread internal disintegration. Brain, liver, and heart were recognizable in the sections. Skin consisted generally of single layer of flattened cells; in small areas were two layers of cells.

The wall of the gestation cyst (ovisac) consists principally of blood clots, superficially of a layer of ovarian stroma of variable width. At its thickest portion the wall has a width of 15 millimeters, at its thinnest point it measures only 3 millimeters. No distinct aperture was recognized. The blood clot portion of the wall includes many long atropic and necrotic chorionic villi. The best preserved villi appear in the superficial nodules previously mentioned. The central region of the thrombus contains more fibrin and the red corpuscles are pale. Less modified blood occurs peripherally. Hæmorrhage progressed radially, producing thus a stratification of the hæmatoma. But no true connective tissue organization had begun in any region.

The stromal portion of the cyst wall is moderately vascular in most regions. Adjacent to the blood clot it is compact and very cellular. Peripherally it is loose and contains much extravasated blood. In the thickest portions the stroma is very vascular. The large venous spaces are generally empty; the arteries are well filled with blood. More peripherally, in certain compact cellular regions, appear also a few more or less flattened, relatively large, graafian follicles with discus proligerus and ovum. A number of primary and atretic ovarian follicles occur in these regions. The ovary retains its peritoneal covering, consisting of a single layer of cells, generally squamous, over certain areas cuboidal.

Fig. 4. Section through wall (lower one-third) of gestation cavity (upper two-thirds). The cavity (dilated graafian follicle) contains centrally two well preserved chorionic villi. The epithelial covering includes a superficial plasmoditrophoblast and an inner cytotrophoblast. Above the villi is a large blood clot, with blood cells largely intact. Below the villi is an epithelial strand of large polyhedral cells, remnants of the membrana granulosa, representing a compressed corpus luteum. The wall of the cyst is covered internally with a thin layer of less modified granulosa cells. The wall consists otherwise of a very vascular ovarian stroma. The section is through the wall of one of the superficial nodules. ×75.

Such of the chorionic villi as are well preserved have the typical structure for the second month of pregnancy. The core of the villus is well vascularized. The covering layer comprises a superficial syncytium (syncytial layer, plasmoditrophoblast) and a subjacent epithelial layer (Langhan's layer, cytotrophoblast), generally complete as a single row of cells (Fig. 5). The nuclei stain well and appear healthy. These villi are characterized by many short, slender, epithelial projections (Fig. 4). Some of these processes have become isolated from the parent villus. In this condition they appear as multinucleated plasmodial masses. In only a few areas do the chorionic villi appear normal. Such areas are in general represented superficially by nodular elevations. Elsewhere the villi, generally long and narrow, and denuded of epithelium, appear atrophic. In addition to chorionic villi and thrombus the cyst wall includes externally an epithelial layer of variable thickness, resting upon the ovarian stroma. The stroma varies greatly in thickness in different regions. It contains a few normal graafian follicles, somewhat compressed, and a number of

Fig. 5. Portion of lining of gestation cyst (region *x*, Fig. 4) showing the character of the cells (persistent membrana granulosa) and the subepithelial connective tissue (theca interna). ×750.

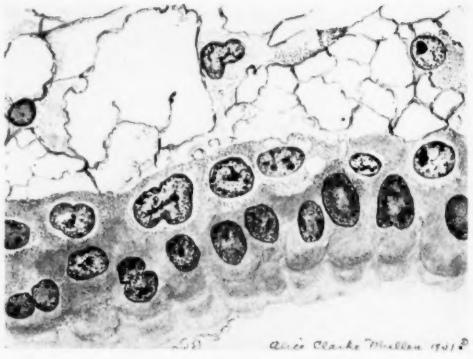

Fig. 6. Portion of the covering of the chorionic villus showing at the left in Figure 4. The superficial nucleated plasmoditrophoblast is clearly distinguishable from the subjacent cytotrophoblast. ×1150.

more or less atrophic primary ovarian follicles. In its thinnest portion the stroma appears to have suffered pressure atrophy. In its thicker portions it contains many approximately normal blood vessels and capillaries.

The cellular covering of the inner border of the stromal portion of the wall is of especial interest. It is of variable thickness. It may comprise only a single layer of cells, with interruptions; the cells are generally elongated and flattened. Where thickest, it may include irregular groups of three or four superimposed polyhedral cells (Fig. 6). This cellular layer rests upon a subjacent, thin, fibrous membrane, suggesting the theca folliculi interna. We are apparently dealing with a portion of the membrana granulosa of the original graafian follicle, which expanded into a gestation cyst following intrafollicular implantation. The cytoplasm of these cells is vacuolated, the condition suggesting solution of fat (lutein) granules.

Of even greater interest is the strand of cells lying between the nearest chorionic villi and the cellular outer boundary of the blood filled cyst. The cells are, in general, large and polyhedral in form (Fig. 7). The nuclei are large, frequently vesicular. The cytoplasm is vacuolated. These cells suggest a corpus luteum. Over certain areas this epithelial layer is closely adjacent to the boundary layer; in general it lies at a variable distance from this layer. Where the cells of these two layers are in juxtaposition the medial cells have transitional characteristics. We are led to interpret the tissue as composed of lutein cells derived from the membrana granulosa. Such interpretation accords with that of Corner (1919) for the origin of the corpus luteum of the pig. Corner has demonstrated that the corpus luteum arises principally from persisting cells of the membrana granulosa, in only small part from cells of the theca interna. In our case there is no evidence of connective tissue constituents, or of ingrowing blood vessels. Bryce, Teacher, and Kerr conclude from their studies that in the human female the corpus luteum arises exclusively from theca cells. This is in agreement with the claim of certain earlier investigators (Koelliker, His, Paladino) but in conflict with that of others of equal authority (Bischoff, Pflueger, Sobotta).

The microscopical data leave no doubt regarding an intrafollicular implantation. As such it seems of considerable importance. There is no evidence here of heterotopic "endometriod" tissue, derivative of displaced parts of Mueller's ducts (Webster, 1904), as described by Sutton (1924) and postulated by him as a prerequisite for ovarian implantation. Haeubner (1928) was unable to detect such tissue and rejects the interpretation.

As previously pointed out by Bryce, Teacher, and Kerr (1908) in their very careful study of an early ovarian pregnancy, the formation of normal chorionic villi within a graafian follicle proves the fetal origin of the covering plasmodium. The only possible other origin of this syncytial layer in our case would be from the membrana granulosa of the ovarian follicle. Though the plasmoditrophoblast of the chorionic villus may at certain areas be confluent with the lutein cells derived from the membrana granulosa, the opposite completely free border is covered with the same type of epithelium and syncytium.

The question, of course, arises whether these epithelial masses lying between the villi

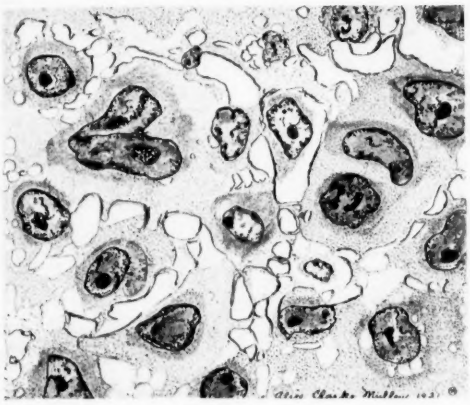

Fig. 7. Portion of the epithelial strand shown in Figure 4 between the chorionic villi and the cyst wall. ×1110.

and the stromal wall should not be interpreted as decidual cells. The question is the more pertinent since previous investigators generally found no sign of decidual reaction within the ovarian stroma. The fact that the cells resemble lutein cells more closely than decidual cells, and especially the conclusion on the part of a number of investigators of the normal origin of the corpus luteum that the lutein cells represent transformed granulosa cells, leads logically to an interpretation of the cells as aborted lutein cells.

In our specimen there occur in certain regions of the ovarian stroma immediately adjacent to the peripheral portion of the hæmatoma, nests of variable but generally small size of large vesicular swollen cells, resembling decidual cells (Fig. 8). The cytoplasm of these cells is moderately acidophilic. The nucleus is generally large and vesicular, occasionally lobulated. Some of these cells contain two nuclei; a few are multinucleated. Similar cell nests have been described by Haeubner, and probably represent the cells interpreted by Sutton as displaced muellerian tissue. Bryce, Teacher, and Kerr also recognized these cells. They admit that they are not unlike decidual cells, and state that "if they be swollen connective tissue elements they would be analogous to the decidual cells in their mode of development, and would represent an effort on the part of the ovarian tissue to react as endometrium does." Since these cells occur

Fig. 8. Portion of ovarian stroma from region adjacent to the gestation cyst. In certain restricted areas the connective tissue cells simulate decidual cells. ×1020.

in relatively small numbers they reject the idea that they constitute anything resembling a real decidua. However, these cells apparently represent a stromal reaction against the invading chorionic villi and the advancing hæmorrhage.

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RELATIONSHIP OF CARCINOMA OF THE BODY OF THE UTERUS AND OF THE OVARIES

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THE purpose of this study is to determine the frequency with which papillary cystadenocarcinoma of the ovaries is associated with carcinoma of the body of the uterus, to review briefly the possible modes of extension from the ovary to the endometrium, and from the endometrium to the ovary, and to record a series of cases in which carcinoma appeared both in the ovary and body of the uterus.

NATURE OF ADENOCARCINOMA OF THE BODY OF THE UTERUS

Adenocarcinoma of the body of the uterus is as a rule a primary lesion, and for a long time confines its growth to endometrium, forming a soft, friable, papillary lesion which is very vascular. Later the growth extends into the muscular wall, and finally penetrates the peritoneum, but its tendency is to localize in the uterine cavity and not to metastasize. Involvement of the lymph nodes and sinuses in cases of carcinoma of the body of the uterus is infrequently seen at operation or necropsy. Cullen, in his study of adenocarcinoma of the body of the uterus, reported only one case of nodal involvement, and he called attention to Winter's series of 44 cases in which there was nodal involvement in only two. This infrequency of nodal involvement has been explained by the fact that the epithelial cells in carcinoma of the body of the uterus are large and do not get into the lymph channels, which are small and few in number. Broders explained this tendency of some carcinomatous cells to stay localized rather than to metastasize as an inherent quality of the cell itself. He observed that cells of adenocarcinoma of the breast and those of melanoepithelioma, metastasize early, whereas, those which are found in the fundus of the uterus, and in the rectum, tend to metastasize late. Invasion from the uterus takes place, usually, by continuous growth toward the bladder, vagina, and rectum, and appears in the form of a solid carcinomatous mass. Graves stated that in operation for carcinoma of the fundus of the uterus, it rarely is necessary to remove the lymph nodes or to perform wide dissection of the parametrium. What is more important than either of these procedures, he wrote, is the removal of the ovaries, on account of the frequent occurrence of implantation metastasis.

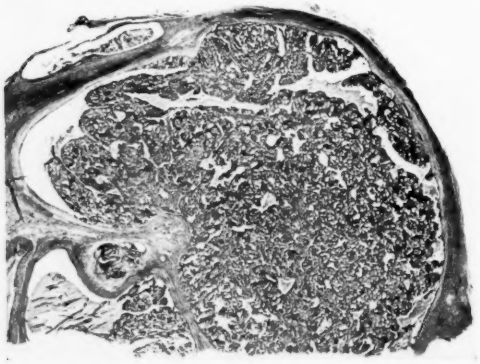

Fig. 1. Papillary cystadenocarcinoma primary in the ovary. (Case 1.)

NATURE OF PAPILLARY CYSTADENOCARCINOMA OF THE OVARY

Papillary cystadenocarcinoma of the ovary tends to be primary in the ovary, and usually starts by degeneration of a benign papillary cystadenoma. This lesion is characterized by a tendency to become bilateral, to rupture early, and to give rise to metastasis. The metastasis takes place by direct extension, or by transplantation, and not by the usual route through the lymph or venous channels. Extensive peritoneal metastasis is seen at operation and at necropsy, and metastatic growths are even felt as direct implantations at a time when the lymph nodes and sinuses are not involved.

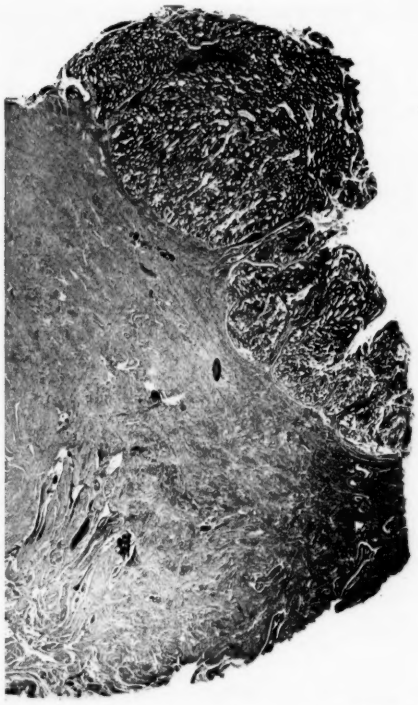

Fig. 2. Papillary cystadenocarcinoma secondary in the endometrium. (Case 1.)

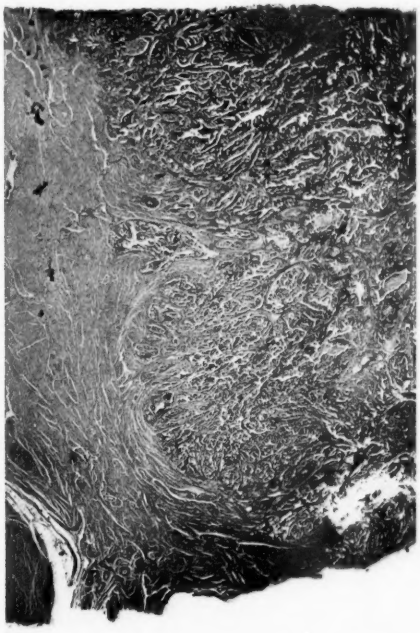

Fig. 3. Primary adenocarcinoma of the body of the uterus. (Case 14.)

MATERIAL STUDIED AND COMMENT

There were 520 cases of carcinoma of the body of the uterus and 616 cases of papillary cystadenocarcinoma of the ovaries in which operation was performed in The Mayo Clinic from 1913 to 1930. In 53 of these cases (Tables I, II, and III) lesions were found in ovaries and uterus. In the cases studied, the uterine lesion was confined to the body of the organ, had not penetrated the uterine wall, and was separated from the ovarian lesion by apparently healthy tissue. Cases in which the condition was extensive and the mode of occurrence was obviously by direct extension, and those cases in which the carcinoma was on the external surface of the uterus only, were not studied in detail, but are included in the tables.

Metastasis in any region of the human body may occur by the lymphatic system, the blood stream, transplantation, or direct extension. It was obviously impossible to determine in most of the cases under consideration the mode by which metastasis occurred, because most of the cases were well advanced, and the pelvis was filled with an extensive carcinomatous process. Furthermore, serial section of the lymphatic structures, the blood vessels, and the fallopian tubes would have

TABLE I

Pathologist's Opinion of Nature of Secondary Growths in Ovary and Fallopian Tube in Five Cases in Which Primary Growth Was in the Uterus

Case	Ovary	Fallopian tube
1	Carcinoma	Carcinoma
2	Carcinoma	No growth
3*	Implants on inside of cysts	No growth
4	Degenerated carcinoma	Adenocarcinoma of fimbria
5	Carcinoma	Involvement of all structures

*The primary growth involved cervix also.

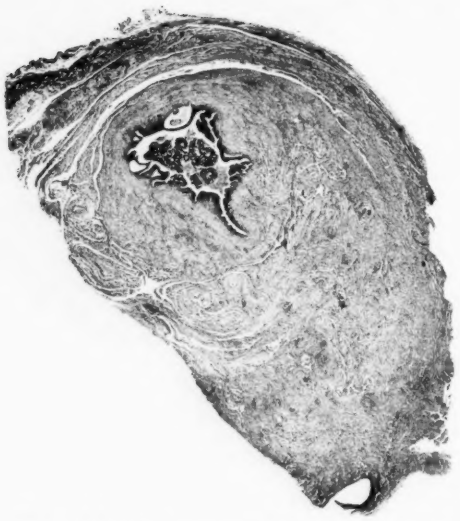

Fig. 4. Adenocarcinoma secondary within the fallopian tube. There was a secondary adenocarcinoma also in the ovary. (Case 14.)

been necessary. However, several cases were studied with the purpose of determining the mode of metastasis. In the cases chosen for study of metastasis the disease was not advanced, and lesions were not seen grossly, except within the ovary and within the uterus, and those lesions were separated by tissue of normal appearance.

In consideration of the possibility of metastasis by the lymphatic system, it was found that the nodes of the parametrium were not involved, and lymphatic structures were free of carcinomatous cells, as far as could be determined by microscopic examination. The lymph vessels of the body of the uterus run between the ovarian artery and the fallopian tube, and then upward to the lumbar nodes. No outlet for the lymph vessels of the body of the uterus has been demonstrated until the lumbar nodes are reached, and here also the ovarian lymph vessels drain. Transmission of material in either direction between the uterus and the ovaries through the lymph vessels, would mean that the material takes a retrograde course. Although retrograde metastasis through the lymphatic channels is a possibility, observers do not feel that it occurs.

TABLE II

Pathologist's Opinion of Nature of Secondary Growths in the Uterus and Fallopian Tube, in Sixteen Cases in Which the Primary Growth Was in the Ovary

Case	Body of uterus	Fallopian tube
6	Carcinoma	No growth
7	Carcinomatous implantations	Carcinoma
8	Carcinoma	Carcinoma of fimbriated end
9	Carcinoma	No growth
10*	Carcinoma	No growth
11*	Carcinoma	No growth
12	Carcinoma, extensive	Carcinoma, bilateral
13*	Carcinoma of right horn	Involvement, bilateral
14*	Carcinoma of endometrium	No growth
15	Papillary adenocarcinoma (implant)	Papillary carcinoma
16*	Extensive implantation metastasis of mucosa and serosa with marked involvement of fundus	Carcinomatous involvement, bilateral
17	Secondary growth	No growth
18	Carcinoma of endometrium and cervix (squamous-cell epithelioma, graded 4, in cervix)	No growth
19*	Carcinoma	No growth
20*	Papillary adenocarcinoma of fundus	Involvement, bilateral
21*	Carcinoma of right side of uterus	Carcinomatous involvement of fimbria, bilateral. Left tube involved also at middle and base

*There were growths in both ovaries, one of which was primary.

The blood vessels of the myometrium in the group of cases of carcinoma of the body of the uterus were free of carcinomatous cells, as well as the blood vessels of the tubes in a few cases that were studied in which the tubes were involved. In general, the blood stream does not seem to be accepted as a means of metastasis of carcinoma.

The normal migration of the ovum from the ovary to the body of the uterus takes place through the fallopian tube. Sampson presented convincing evidence that migration of cells also takes place in the reverse direction (Figs. 1, 2, and 3) from the uterus to the ovary, and he demonstrated endometrial and carcinomatous cells in the ovary and peritoneal cavity that had originated in the uterus. In several of my cases carcinomatous cells were found in the lumen of the fallopian tube, and the blood vessels and lymphatic channels of

TABLE III

Pathologist's Opinion of Nature of Growths in Uterus, Ovaries, and Fallopian Tubes in Thirty-one Cases in Which the Situation of the Primary Growth Could Not Be Determined*

Case	Uterus	Ovary	Tube
22	Adenocarcinoma of body	Multilocular carcinomatous cystadenoma	No growth
23	Carcinoma of body and cervix	Intracystic carcinomatous cystadenoma	No growth
24	Carcinoma of body	Intracystic carcinomatous cyst	No growth
25	Carcinoma of body	Bilateral carcinomatous cystadenoma	No growth
26	Carcinoma of body involving whole of uterus	Bilateral intracystic papillary carcinomatous cystadenoma	No growth
27	Carcinomatous uterine polyps; papillary cystadenoma	Intracystic and extracystic papillary carcinomatous cystadenoma	No growth
28	Carcinoma of body with involvement of cervix	Bilateral carcinomatous papillary cystadenoma	No growth
29	Advanced carcinoma of body	Intracystic and extracystic carcinomatous cystadenoma	Secondary carcinoma
30	Adenocarcinoma of uterus involving cervix	Adenocarcinoma	No growth
31	Extensive carcinoma of body	Carcinoma	No growth
32	Carcinoma of body	Intracystic carcinomatous papillary cystadenoma	No growth
33	Carcinoma of fundus	Bilateral carcinomatous cystadenoma	No growth
34	Carcinoma of body	Bilateral infected carcinomatous cystadenoma	No growth
35	Carcinomatous portions in endometrium	Carcinomatous papillary cystadenoma	No growth
36	Carcinomatous mass, involving body of uterus	Bilateral intracystic and extracystic papillary carcinomatous cystadenoma	No growth
37	Early carcinoma of body	Intracystic and extracystic papillary carcinomatous cystadenoma	No growth
38	Carcinoma of body	Bilateral cystadenoma with region of carcinoma	Fimbriated end of right fallopian tube incorporated in carcinoma of right ovary
39	Adenocarcinoma of body	Multilocular intracystic adenocarcinoma	No growth
40	Adenocarcinoma involving entire fundus	Intracystic papillary carcinomatous cystadenoma	No growth
41	Carcinomatous polyp of body	Carcinoma	Carcinomatous portion in lumen
42	Carcinoma of fundus	Solid carcinomatous cystadenoma	No growth
43	Carcinoma of body	Bilateral carcinomatous cystadenoma	Involvement of right tube
44	Adenocarcinoma of body	Adenocarcinoma	No growth
45	Annular papillary carcinoma of body	Intracystic and extracystic carcinomatous papillary pseudomucinous cystadenoma; solid carcinoma	No growth
46	Papillary carcinoma of body	Bilateral degenerating papillary carcinoma	No growth
47	Adenocarcinoma of fundus	Bilateral carcinomatous papillary cystadenoma	No growth
48	Adenocarcinoma of body	Bilateral adenocarcinoma	No growth
49	Adenocarcinoma of body	Bilateral cyst containing carcinoma	No growth
50	Diffuse adenocarcinoma of body	Intracystic papillary adenocarcinoma	No growth
51	Diffuse papillary adenocarcinoma; also, adenocarcinomatous polyp of body	Multilocular solid intracystic papillary adenocarcinoma	No growth
52	Papillary adenocarcinoma of body	Adenocarcinoma	No growth

*In one case, which is not noted in any of the tables, there was a carcinoma of the body of the uterus, which apparently was independent of the carcinomatous cystadenoma of the ovary.

the tube were apparently normal. Clark and Norris referred to 2 cases in a series of 115 in which carcinomatous cells were found in the lumen of the tube. They expressed the belief that transtubal dissemination occurs in many cases of combined ovarian and uterine carcinoma. In another report of 101 cases of carcinoma of the fundus Norris reported 8 cases in which the ovaries were involved, and Cameron reported 9 cases of carcinoma of the ovary secondary to carcinoma of the uterus in a series of 31 cases. It was not possible in every case of my study to find in the tube proof that the carcinomatous tissue had passed through, but, in 15 of the 53 cases, secondary involvement of the tube was present. It is possible that dislodged carcinomatous cells may pass through the lumen and not become attached until they reach the site of the future secondary growth.

It was likewise difficult to decide, in these cases, which was the primary and which was the secondary lesion. The size of the tumor was not conclusive evidence, for a secondary growth may be larger than a primary growth. The symptoms of carcinoma in both organs are similar. Carcinomata of the body of the uterus probably give rise to symptoms earlier than do lesions in the ovary; metrorrhagia may occur early in the presence of carcinoma of the uterus, whereas carcinoma of the ovary often does not produce symptoms until a palpable tumor, or pain, is noticed. In this study, the chronologic appearance of symptoms was of little value. The study of the cell gave more information. MacCarty has stated that study of the fresh tissue cell may give information regarding the origin of the carcinoma. The microscopic study made of fresh tissue in this group of 53 cases, revealed 5 cases of primary uterine carcinoma with metastasis to the ovary (Table I), 16 cases in which the lesion was primary in the ovary and secondary in the uterus (Table II), 31 cases in which it was impossible to determine the situation of the primary lesion (Table III), and 1 case in which there was a carcinoma in the uterus and another in the ovary, each independent carcinomata (see footnote on Table III).

SUMMARY

In a series of 520 cases of adenocarcinoma of the body of the uterus, there was associated carcinoma of the ovary in 11.9 per cent, and in 616 cases of papillary cystadenocarcinoma of the ovary, there was associated carcinoma of the body of the uterus in 8.6 per cent. The fallopian tube should be considered as one of the means through which transplantation takes place. Because of the similarity of the adenocarcinomatous cells in the embryologically similar tissues, it is often difficult to determine which carcinoma is primary and which is secondary and whether or not there may originally have been two independent carcinomata. In cases of carcinoma of the ovaries, the possibility of metastasis to the uterine endometrium, even when there is no gross peritoneal evidence of extension of the malignant growth, must be borne in mind. Because of this possibility, hysterectomy at the time of removal of the ovaries must be seriously considered.

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ABDOMINAL AND PELVIC FASCIAS WITH SURGICAL APPLICATIONS

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TO visit clinics and to read descriptions of various operative procedures, one naturally must resort to a textbook on anatomy to be able to visualize those structures which are referred to so freely and apparently so understandingly by the various authors and operators; but to one's bewilderment, even the best American and English textbooks do not satisfactorily discuss this anatomy which we are anxious to understand more intelligently.

Various German authors: Halban, Veit, Tandler, Martin, and, in America, Gallaudet, agree that there are two systems of fascias in the abdominal and pelvic regions; the one, a thick fibrous sheet which covers the voluntary muscles, such as the diaphragm, the oblique muscles, the transversalis, the obturator internus, the ileo-psoas, and the pyriformis.

Various daughter splits may be given off from the parent fascia, but being derived from fascia covering voluntary muscles, the daughter fascia will surround voluntary muscles and not an involuntary muscle, such as the vaginal tube which is mistakenly supposed to be surrounded by a definite fascial layer, such as surrounds the deep transverse perineal muscle. Involuntary muscles and abdominal organs are surrounded by a structure which resembles the superficial fascia of the abdomen more than it does the deep fascia of the abdomen. This layer is referred to by the German authors as the connective tissue fascia (Bindegewebe) and by Gallaudet as the "Subperitoneal fibro-areolar layer (Fig. 1, *S.P.*)."

The abdominal wall may be visualized as consisting of a cylindrical wall of voluntary muscle, the oblique and transversalis muscles. The muscle layer is surrounded by a distinct sheath of fibrous tissue. The superficial layer of this fibrous sheath is referred to as the *deep fascia of the abdomen* (Fig. 1, *D.F.*) and this structure together with the tendon of the external oblique muscle is the layer upon which our attention is directed in a repair of the abdominal wall. The deeper layer (Fig. 1, *T.F.*) is referred to as the *transversalis fascia* and is too often disregarded in a repair of the abdominal wall, except in the upper quadrants, where it becomes more conspicuous due to the hypertrophied deep layer of the internal oblique muscle tendon which fuses with the tendon of the transversalis muscle and with the transversalis fascia. It thereby becomes more conspicuous and less likely to be ignored, particularly if the external oblique tendon is attenuated. Superficial to the so called deep fascia of the abdominal wall is the superficial fascia (Fig. 1, *S.F.*), and deep to the transversalis fascia is a layer not unlike the superficial fascial layer, "the subperitoneal fibro-areolar layer" (Fig. 1, *S.P.*). Superficial to the superficial fascia is the skin and deep to the subperitoneal fibro-areolar layer is the peritoneum. It can then be seen that the central layers of the abdominal wall are the voluntary muscles which are covered superficially and deeply by a definite fascial layer. Superficial to the superficial sheath (Fig. 1, *S.F.*) and deep to the deep sheath (Fig. 1, *S.P.*) is a layer of fibro-areolar tissue which transmits the nerve and blood supply of the abdominal wall.

Superficial and deep to these fibro-areolar layers are the skin and the peritoneum, respectively. In general, the skin and the peritoneum, the subcutaneous and the subperitoneal layers, the deep and transversalis fascias, correspond except that the cutaneous and subcutaneous layers are of a coarser texture than the corresponding peritoneal and subperitoneal layers; which may be due to external influences.

The subperitoneal (Fig. 1, *S.P.*) and subcutaneous (Fig. 1, *S.F.*) layers consist of two portions, a fibrous and an areolar layer in which there are varying quantities of adipose tissue. In the subcutaneous region the fatty

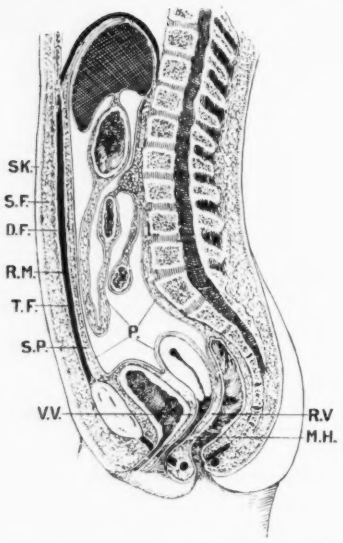

Fig. 1. Diagrammatic sagittal section of the abdomen and pelvis. To show the layers of the abdominal wall and the subperitoneal tissue surrounding the abdominal and pelvic organs and structures. *S.K.*, Skin; *S.F.*, superficial fascia; *D.F.*, deep fascia; *R.M.*, rectus muscle; *T.F.*, transversalis fascia; *S.P.*, subperitoneal fibro-areolar layer; *M.H.*, hæmorrhoidal mesenteroid of vagina; *V.V.*, vesicovaginal area; and *R.V.*, rectovesical area.

layer is referred to as Camper's layer, while the deeper fibrous layer is called Scarpa's layer. We will first consider the voluntary fascial layer which ensheathes the transversalis muscle medially (Fig. 1, *T.F.*), and, secondly, the subperitoneal fibro-areolar layer (Fig. 1, *S.P.*), in which the various abdominal and pelvic organs and structures develop.

VOLUNTARY FASCIAL LAYER

The transversalis fascia is the term applied to the deep layer of fascia which ensheathes the voluntary muscles on the medial surface of the abdominal wall (Fig. 1, *T.F.*). Not only is it applied to that layer which covers the transversalis muscle medially, but also the abdominal fascial layer which surrounds the diaphragm. It can be traced distally as the sheath of the ileo-psoas muscle to the false pelvis (Fig. 2, *I.F.*), it enters into the true pelvis where it covers the inner surface of the body of the pubis (Fig. 2, *P.*) and the obturator internus muscle (Fig. 2, *O.T.F.*). In the concavity of the sacrum it ensheathes the pyriformis muscle and at the arch of the pubis (Fig. 2, *A.C.*), it continues distally as the fascia covering the adductor muscles of the thigh.

From the parent fascia, daughter layers are given off which ensheathe voluntary muscles. Such a splitting occurs in the pubic arch (Fig. 2, *A.C.*), as the fascia covering the obturator internus muscle proceeds to become the fascia of the adductor group of muscles in the thigh. A double split is given off at each ramus which is directed medially and fuses with the opposite splits to form the so called superficial and deep layers of the triangular ligament (Fig. 2, *T.L.*), while the voluntary muscle contained therein, is called the deep perineal or triangularis muscle.

Along a line running from the posterolateral surface of the body of the pubis to the spine of the ischium (Fig. 2, *W.L.*), the obturator portion of the transversalis fascia sends two daughter splits medially and distally to ensheathe the levator ani muscle. The muscle arises from the posterior surface of the pubis and from the angle between the two daughter splits at their junction with the obturator fascia. The insertion of the levator is into a median raphé which extends from the tip of the coccyx to the anus (Fig. 2, *C.A.*). It also surrounds the anus laterally and sends a small strand of fibers into the perineal body. As the pubococcygeal portion passes lateral to the vagina and the urethra it sends few small muscular strands into the lateral wall of the vagina and a small bundle medially between the vagina and the urethra. Just as the fibers posterior to the anus and posterior to the vagina act as voluntary constrictors of these structures so the transverse fibers between the urethra and the vagina may aid as voluntary constrictors of the urethra.

The daughter split of fascia, which ensheathes the levator ani on its proximal or visceral surface (Fig. 3, *V.L.*), is referred to as the visceral or deep layer of the levator fascial sheath; while the split which encloses the levator ani on its inferior or superficial surface (Fig. 3, *P.L.*) is referred to as the parietal or superficial layer of the levator sheath. The coccygeus muscle, arising from the spine of the ischium, inserts into the lateral border of

the coccyx and does not act as a levator muscle other than through its action on the coccyx. The pyriformis muscle aids the coccygeus in completing the muscular outlet of the true pelvis posterior to the levator ani muscle.

In the perineal body, anterior to the anus, unlike on the lateral and posterior areas of the anus, the muscular and fascial layers are not distinct and thereby not readily identified; for there is a blending of the fascial layers with transverse muscular strands of the levator muscle, together with the layers of the triangular ligament and the deep perineal muscle, contained therein.

INVOLUNTARY FASCIAL LAYER

The involuntary fascial layer (Fig. 1, *S.P.*), being subperitoneal and deep to the fascia covering the voluntary abdominal muscles, corresponds to the subcutaneous fibro-areolar layer (Fig. 1, *S.F.*), which lies superficial to the fascia covering the voluntary abdominal muscles. It is composed of a fibrous and an areolar layer which contains in its meshes varying quantities of adipose tissue. The superficial organs such as the sweat glands, the sebaceous glands, the hair follicles, and the breast, originating from the epithelial layers of the true skin, penetrate the subcutaneous tissue and mature. In addition, the subcutaneous tissue serves as a path for the passage of arteries, veins, nerves, and lymphatics of the superficial abdominal wall, and its contents. Just as the organs related to the superficial structures develop in the subcutaneous fibro-areolar layer, so the organs and structures related to the abdominal and pelvic regions develop in the corresponding subperitoneal fibro-areolar layer. Superficially, the occlusion of the duct of a sebaceous gland may cause the gland to enlarge and distort the surface of the skin, but in doing so the subcutaneous tissue becomes so attenuated that the gland appears to be covered only by the skin. In the same manner the organs which develop in the subperitoneal fibro-areolar layer increase in size and may force the peritoneum before them and bulge into the peritoneal cavity. In the case of the intestines, they grow into the peritoneal cavity so completely that they can be said to be pedunculated and apparently lose their connection with the subperitoneal fibro-areolar layer, except through their mesentery and the very thin fibro-areolar layer which surrounds them. Such organs as the liver, spleen, intestines, uterus, and fallopian tubes, which are considered to be intraperitoneal, by their growth and bulging into the peritoneal cavity, lose most of their fibro-areolar covering; while those structures such as the kidney, ureter, the distal portion of the bladder, the vagina and the rectum, which do not bulge intraperitoneally, are covered by a very definite layer of fibro-areolar tissue. This readily recognizable layer in the region of the kidney resembles Camper's layer of the subcutaneous tissue and is often referred to as the fatty capsule of the kidney. Peripheral to the fatty layer there is a more concentrated layer which resembles Scarpa's layer of the subcutaneous tissue.

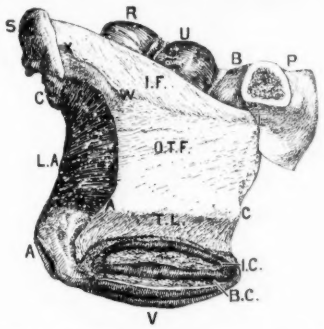

Fig. 2. To show continuation of transversalis fascia into the true pelvis. Specimen was obtained after removing all of the ileum, and the ischium and the distal portion of the pubic bones without disturbing the transversalis fascia, on the medial surface of the pelvic bones. *S.*, Sacrum; *C.*, coccyx; *L.A.*, levator ani; *A.*, anus; *R.*, rectum; *U.*, uterus; *B.*, bladder; *P.*, body of pubic bone; *I.F.*, iliac fascia; *O.T.F.*, obturator internus fascia; *T.L.*, triangular ligament; *I.C.*, ischiocavernosus muscle; *B.C.*, bulbocavernosus muscle; *V.*, vaginal introitus.

That the intestines are still covered by this layer of tissue, even though very thin, is evident by the tendency to fatty deposits in it, such as the appendices epiploicæ. That the uterus, itself, is covered by this layer is more evident during pregnancy, when there is a hypertrophy of this tissue. It is evidenced by our ability to separate the peritoneum from

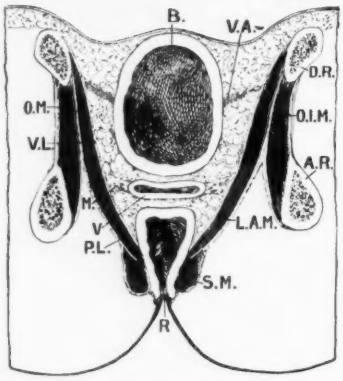

Fig. 3. Horizontal section of the true pelvis. To show the pelvic continuation of the transversalis fascia where it ensheathes the voluntary levator and obturator internus muscles. The bladder and the vagina being involuntary muscles are surrounded by subperitoneal fibro-areolar tissue in which there are varying quantities of fatty deposits. Between the vagina and the bladder and between the vagina and the rectum, there is little or no fatty deposit and very little fibrous tissue, consequently the tissue connecting the bladder to the vagina and also the rectum to the vagina is merely an areolar tissue. *O.M.*, Obturator membrane; *V.L.*, visceral layer of levator fascia; *P.L.*, parietal layer of levator fascia; *M.*, mesenteroid of vagina; *V.*, vagina; *R.*, rectum; *S.M.*, sphincter muscle; *B.*, bladder; *V.A.*, vesical artery; *L.A.M.*, levator ani muscle; *D.R.*, descending ramus of pubic bone; *A.R.*, ascending ramus of pubic bone; and *O.I.M.*, obturator internus muscle.

the lower half of the uterus, as is practiced in the low flap cæsarean section. Summarizing, one notes that those organs which are considered to be intraperitoneal due to their growth into the peritoneal cavity, are covered by a very thin layer of fibro-areolar tissue, while the extraperitoneal structures and organs are covered by a more definite layer of fibro-areolar tissue in which there is a greater deposit of adipose tissue; accompanying blood vessels there is a concentration of the fibrous portion which suggests a definite ligamentous structure. On those areas which are compressed by expansion of the organ or structure there is an increased deposit of adipose tissue in the areolar portion.

The intraperitoneal and extraperitoneal structures of interest gynecologically are the bladder, rectum, vagina, and uterus.

Bladder. Practically the entire collapsed bladder (Fig. 3, *B.*), except its abdominal surface, is surrounded by a thick layer of fibro-areolar tissue, which, as in the region of the kidney, can be readily separated from the bladder, except in the regions of the superior, middle, and inferior vesical vessels. On the abdominal surface of the bladder the fibro-areolar layer is so thin that the peritoneum appears to be intimately connected to the bladder, and for this reason does not permit complete extraperitoneal mobilization of the organ. The attenuation of the fibro-areolar layer subperitoneally must be remembered when performing the extraperitoneal cæsarean section as is described by Latzko. To the concentration of the fibrous portion around the vessels supplying the bladder, the term, true ligaments of the bladder, has been applied.

On the vaginal surface of the bladder (Fig. 1, *V.V.*) there is a thinning of the fibro-areolar layer, but there is no fusion to the corresponding surface of the vagina, because it likewise is covered by a similar thin layer of fibro-areolar tissue. These opposing layers are held together by a very loose areolar tissue, which, because of its spider-like texture, can be readily broken and give the appearance of a vesicovaginal space. A similar condition exists between the posterior wall of the vagina (Fig. 1, *R.V.*) and the anterior surface of the rectum, to which the term rectovaginal areolar area has been applied.

Rectum. The rectum, being extraperitoneal, is surrounded by a very heavy fibro-areolar layer in which there is an extra deposit of fatty tissue. The inferior hæmorrhoidal artery, being a branch of the internal pudendal artery, supplies that portion of the rectum distal to the levator ani muscle (Fig. 3, *L.A.M.*) and consequently does not lie in the subperitoneal tissue. The superior hæmorrhoidal and the middle hæmorrhoidal vessels traverse the fibro-areolar layer and are surrounded by a denser fibro-areolar tissue. The many ramifications of the superior hæmorrhoidal artery together with its thickened fibrous surroundings form very definite supporting strands of fibrous and vascular tissue which prevent the prolapse of the rectal wall, but not of the mucous membrane.

Muellerian ducts. The muellerian ducts (Fig. 4) being retroperitoneal and covered by

fibro-areolar tissue, are located posteriorly and lateral to the midline. They receive their blood supply independently. Each duct can be likened to a loop of intestine with its nerve and blood supply entering it at a fixed border. There is the usual thickening of the fibro-areolar layer around the vascular supply, which terminates in the lateral wall of the entire tube, from its proximal to its distal extremity. A similar structure attached to an intraperitoneal organ is called a mesentery and to have an accurate comprehension of this structure and yet to differentiate it from a true mesentery, the term mesenteroid may be applied. As the embryo matures, the distal portion of the two ducts approach each other and fuse at the border opposite the mesenteroid attachment. Further development results in the absorption of the intervening septum, with the formation of a single cavity. The proximal portion of the single cavity becomes the uterus and the distal portion the vagina, while the intervening portion becomes the cervix. The uterus and the vagina lie in the center of a sling (Fig. 5), the lateral support being the individual mesenteroid to that half of the reproductive apparatus. As the uterus and the fallopian tube develop, they bulge intraperitoneally and draw their blood supply with them, this thereby angulates the uterine artery but does not change the direction of the vaginal arteries.

Pushing the peritoneum before it in the midline, together with the fixation of the ovarian extremity of the fallopian tube, causes the formation of the so called lateral or broad ligaments of the uterus (Fig. 5, *B.L.U.*). The leaves of the broad ligaments can be separated from each other due to the presence of a small amount of fibro-areolar tissue enclosed between them. The adult uterus, being intraperitoneal, appears to be devoid of an extraperitoneal fibro-areolar layer, but during pregnancy this layer hypertrophies, particularly in its lower half, and permits the performance of the so called low flap cæsarean section, in which the peritoneum of the lower half of the anterior surface of the uterus is mobilized before the uterus is incised.

Vagina. The vagina, remaining extraperitoneal, is covered by a layer of fibro-areolar

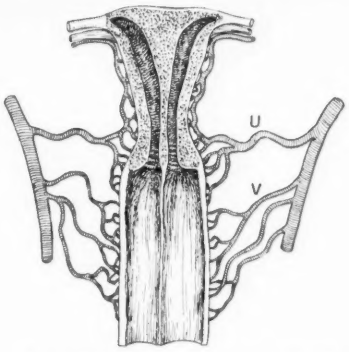

Fig. 4. Septate uterus and vagina with blood supply to each individual portion. To show reproductive apparatus in center of sling partly formed by blood vessels. *U.*, Uterine artery and *V.*, vaginal arteries.

tissue, which is most pronounced along its lateral border (Fig. 5, *B.L.V.*) where the vaginal portion of the uterine and the vaginal arteries traverse the space between the lateral border of the vagina and the superior layer of the levator fascia (Fig. 3, *M.*). There is merely a spiderweb-like attachment which unites the vagina to the bladder anteriorly and to the rectum posteriorly. It is possible readily to separate the vagina from the bladder and from the rectum by incising the involuntary muscular wall of the vaginal tube and by breaking the fine areolar connection which binds the vagina to the vaginal surface of the bladder or to the vaginal surface of the rectum.

Many operators ignore the presence of a very delicate areolar connection between the rectum and the vagina, and between the bladder and the vagina. They refer to this area as the rectovaginal and the vesicovaginal space because of the ease with which this area is entered during plastic operations (Fig. 1, *V.V.* and *R.V.*).

The rectovaginal area unlike the vesicovaginal area is divided into an upper four-fifths and a lower one-fifth by a distinct horizontal mesenteroid (Fig. 1, *M.H.*). This is formed by the terminal portion of the middle hæmorrhoidal arteries and veins together with their fibrous surrounding, as they pass from the rectum to the posterior surface of the vagina, about 2 centimeters from the triangular ligament. The ligamentous appearance of

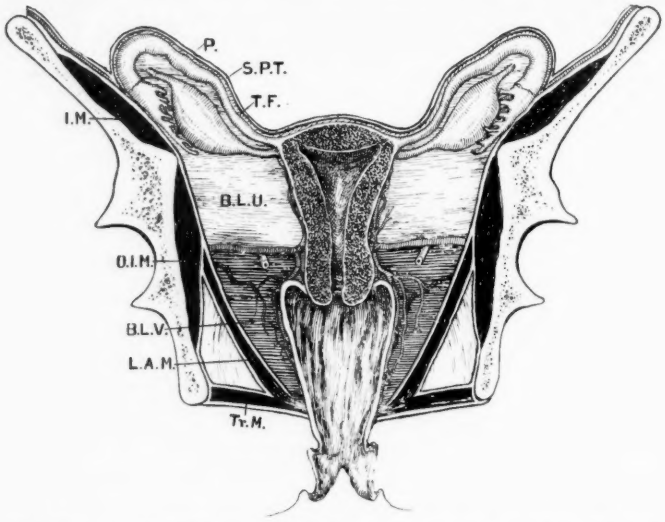

Fig. 5. Diagrammatic lateral sagittal section of true and false pelvis. To show continuation of transversalis fascia into the pelvis where it ensheathes the voluntary, iliacus, the obturator internus, the levator ani and the deep transverse perineal muscles. The reproductive apparatus lies in the subperitoneal fibro-areolar tissue which forms definite lateral supports for the uterus and the vagina. *P.*, Peritoneum; *S.P.T.*, subperitoneal fibro-areolar tissue; *T.F.*, fallopian tube; *I.M.*, iliacus muscle; *O.I.M.*, obturator internus muscle; *B.L.V.*, broad ligament of vagina (lateral mesenteroid of vagina); *L.A.M.*, levator ani muscle; and *Tr.M.*, triangularis muscle.

this structure has caused it to be called the pillar of the rectum. It has been utilized in operations for rectocele, whereby the vaginal termination of the mesenteroid is divided and reattached on the posterior wall of the vagina proximal to its original termination.

THE LATERAL OR BROAD LIGAMENTS

Stretching from the fallopian tubes, proximally, to the triangular ligament, distally, lateral to the uterus and the vagina, there is a distinct plane through which the uterine and vaginal vessels pass to their termination (Fig. 5). The vessels are surrounded by a thickening of the subperitoneal fibro-areolar tissue, which is much heavier extraperitoneally than it is intraperitoneally. That intraperitoneal portion which stretches from the uterus to the lateral pelvic wall has been called the lateral or broad ligament of the uterus (Fig. 5, *B.L.U.*).

While the extraperitoneal portion, which is less accessible, seems to be less familiar and consequently unnamed (Fig. 5, *B.L.V.*), the proximal portion of the extraperitoneal ligament or mesenteroid has been recognized and called the cardinal ligament. It must be understood that the cardinal ligament is not a solitary thickened band which stretches from the axis of the uterus to the lateral wall of the pelvis, but as the proximal border of a trapezoid ligament or mesenteroid which extends from the axis of the uterus to the triangular ligament and stretches from the lateral border of the vagina to the superior layer of the levator of the levator fascia. Just as the broad ligament of the uterus prevents the passage of the hand from the uterovesical area to the uterorectal area, so the lateral mesenteroid of the vagina (Fig. 3, *M.*) prevents the lateral passage of the finger from the vesicovaginal area to the rectovaginal area. It thereby prevents lateral mobilization of the vagina.

Comparing the lateral mesenteroid of the vagina (Fig. 5, *B.L.V.*) with the lateral ligament of the uterus (Fig. 5, *B.L.U.*), we find that the

latter is much wider and thinner than the former, which is narrower and thicker due to the vaginal plexus with its thickened fibro-areolar concentration. Poor fascial structure will cause a greater relaxation of the uterine portion than of the vaginal portion and thereby permit abnormal mobility of the uterus.

SHAPE OF THE UTERUS, CERVIX, AND VAGINA

The normal uterus and the vagina are flattened anteroposteriorly and elongated laterally. The anteroposterior flattening of the vagina is not due entirely to the pressure from the bladder anteriorly and the rectum posteriorly, but a very important contributing factor is the lateral attachment of its mesenteroid to the superior layer of the levator fascia.

The uterus being likewise suspended in the center of a sling formed by the broad ligaments, will conform to stress and strain and become slightly wider in its lateral dimension and correspondingly narrower in its anteroposterior dimension.

The cervix, which bulges freely into the vault of the vagina, has no lateral attachment and consequently assumes a cylindrical form.

RETROVERSION

The broad ligament of the uterus extends laterally as a relatively thin structure; but in the region of the union of the body of the uterus with the vaginal vault, where the uterine vessels traverse the lateral ligament, the structure of the broad ligament changes abruptly. This thickening is sometimes referred to as the cardinal ligament and is functionally the horizontal axis of the uterus. The lateral attachment of the uterine broad ligament, which is anterior to the long axis of the vagina, will throw the uterus anteriorly and thereby cause it to be further anteverted by abdominal pressure. Normally retroversion is prevented by the uterosacral muscles which are inserted distal to its axis, the cardinal ligaments; and also by the round ligaments or more properly the round muscles of the uterus, which are inserted proximal to its axis. To a lesser degree the uterovesical fold of peritoneum aids the round and uterosacral muscles in preventing that position.

In those cases in which there is a relaxation of the broad ligament, due either to a lateral attachment which is posterior to the normal attachment, or to an inherent loss of elasticity of the structures; intra-abdominal pressure may overcome the stress of the round and uterosacral muscles with the resulting retroversion of the uterus. For the repair of this condition we have been unable to transplant the lateral attachment of the broad ligament anteriorly, but Dr. Bissell has satisfactorily plicated the relaxed broad ligament in addition to the usual shortening procedures upon the round and uterosacral muscles. The writer is of the opinion that of all other operative procedures, the method described by Simpson for the cure of retroversion is perhaps the next satisfactory. However, it does not utilize the uterosacral muscles nor the lateral attachments of the uterus, but the round muscles are at least restored in their normal anatomical position.

PROLAPSE

The anatomical difference between the broad ligament of the uterus and the corresponding mesenteroid of the vagina would lead one to assume that a subnormal fascial tissue would cause a greater degree of relaxation in the thin uterine portion than in the thick, narrow, vaginal portion. Consequently a prolapse of the vagina is a relatively rare condition, while a prolapse of the uterus is comparatively frequent. The relaxation permits a retroversion which later descends, especially in those cases in which the cardinal ligament is attached to the vault of the vagina, rather than to the uterovaginal area. The normal attachment of the cardinal ligament to the uterovaginal area may account for the few cases of prolapse compared to the many cases of retroversion; but in a decade or more the cardinal ligament may relax sufficiently to permit a prolapse of the retroverted uterus.

For the early case of prolapse, operation. For retroversion together with a tightening of cardinal ligaments, as described by Alexandroff, may suffice.

HYSTERECTOMY

Whenever the uterus and the cervix are removed and the vault of the vagina is closed

anteroposteriorly, we note relatively frequently several months following the operative procedure that there is a slight relaxation of the newly constructed vault of the vagina. By approximating the medial attachments of the lateral ligaments in the midline, which results from a lateral-to-lateral closure of the vault of the vagina, a tenser sling will be formed which may be sufficient to prevent a prolapse of the vault of the vagina in those cases which are prone to a general fascial relaxation. Dr. Kennedy has utilized this mechanical principle in his report of a series of total hysterectomies. Other than for ease in peritonealization, the attachment of the divided round muscles to the vault of the vagina is unnecessary, because the lateral ligament of the vagina is a much more efficient support in preventing prolapse than the thin strand of round muscle.

In closing the vault of the vagina following a vaginal hysterectomy, the approximation of the cardinal ligaments in the midline, by a side-to-side closure, will construct a deeper and more anatomical condition of the vagina than will result from procedures in which the cardinal ligaments are not approximated medially.

CYSTOCELE

That portion of the female reproductive system known as the vagina resembles the proximal portions of the genital system in that it is composed chiefly of involuntary muscular fibers together with fibrous tissue, and like the proximal portion it is surrounded by a fibro-areolar layer of tissue which is heaviest laterally where it aids in the formation of the vaginal mesenteroid, while anteriorly and posteriorly the covering of fibro-areolar tissue is almost negligible. To repair the so called cystocele, which is probably a relaxation of the anterior wall of the vagina rather than a distinct laceration of it, the repair is directed to the wall of the vagina, which, like in the gastro-intestinal tract, is composed of an inner circular and an outer longitudinal layer of involuntary muscular fibers, and not to a definite fascial layer which lies peripheral to the muscular layer, such as surrounds voluntary muscle. There is, however, the thin fibro-areolar layer which surrounds involuntary muscle but as such it can not effectively be used alone in the cure of cystocele.

RECTOCELE

The posterior wall of the vagina, like the anterior wall, is composed of a muscular wall which is surrounded by a thin layer of fibro-areolar tissue. Posteriorly and laterally the rectum is surrounded by a heavy layer of fibro-areolar tissue in which there is a large deposit of fat, but anteriorly the fibro-areolar layer is attenuated and corresponds to the covering on the posterior wall of the vagina. A delicate areolar tissue connects these opposing coverings, but readily permits mobilization of the vagina from the rectum, except at the junction of the upper four-fifths with the lower one-fifth, where there is the usual thickening around the vaginal branches of the middle hæmorrhoidal vessels as they pass from the anterior surface of the rectum to the posterior surface of the vagina. The term, hæmorrhoidal mesenteroid of the vagina, may be applied to this structure which is utilized by Ward in his operation for rectopexy, whereby the vaginal attachment of the mesenteroid is divided and transplanted proximal to its normal position.

PERINEORRHAPHY

No plastic repair is complete without including the perineal body, which is composed of a voluntary muscle ensheathed by a very definite fascial layer. Not only does the deep triangular muscle together with the superficial and deep layers of the triangular ligament, enter into the formation of the perineal body but there are also a few fibers from the levator ani, which branch medially from the pubococcygeus, together with the voluntary fascial covering them, which aid in the formation of the conglomerate fibromuscular body. In a nullipara, the pubococcygeus can be felt as a very definite pillar lateral to the vagina about 2 centimeters from the hymen, but of this heavy bundle only a few fibers enter into the perineal body. To deliberately draw the pubococcygeus into the perineal body is unanatomical but it may improve the tone of the levator muscle. The tightening of the triangular ligament indirectly increases the tone of the lat-

eral mesenteroid of the vagina by fixing its distal and medial extremity. Indirectly by repairing the anterior and posterior walls of the vagina and the lacerated or relaxed perineum, one not only improves the cystocele, the rectocele, or the relaxed perineum but the general tone of the pelvic outlet is increased, due to the tightening of the relaxed lateral mesenteroid of the vagina which extends from the lateral wall of the vagina to the superior layer of the levator fascia with which it is intimately connected.

CERVICAL REPAIR

To amputate the vaginal portion of the cervix should not be a dangerous procedure unless the amputation is so high as to expose the lateral attachment of the uterus and thereby damage one or more of its contained blood vessels. Sturmdorf in making a cervical cuff is certain that the mobilization is done anteriorly and posteriorly and not laterally, where danger is encountered.

In postpartum examinations of the cervix for a possible laceration, traction on the anterior and the posterior lips of the cervix will add to the already greatly distorted condition, because traction is made on the most elastic portion which is not efficiently transmitted to the body of the uterus. Traction laterally, however, will be more efficient due to the attachment of the broad ligament of the uterus.

POSTPARTUM HÆMORRHAGE

In all postpartum inspections for hæmorrhage, the sulcus of the vagina must not be overlooked, particularly since it is the least elastic portion of the vagina, and connects with the vaginal plexus contained in the lateral mesenteroid.

SUMMARY

1. Two systems of fascia are found in the abdominal and pelvic regions, the one a thick fibrous sheet which ensheathes voluntary muscle and the other a fibro-areolar layer which surrounds involuntary muscle and organs.

2. Organs and structures related to the skin develop in the subcutaneous layer, while organs and structures related to the abdominal and pelvic cavities develop in the subperitoneal layer.

3. Areas subjected to pressure by distention of the organ or structure are protected by an increased deposit of adipose tissue in the areolar layer.

4. Vessels traversing the fibro-areolar layer are surrounded by an increase of the fibrous tissue.

5. The mesenteroid to each muellerian duct forms the lateral support of the adult uterus and vagina.

6. The vagina like the rectum is a muscular tube composed of an inner circular and an outer longitudinal layer of involuntary muscular fibers. It is covered by fibro-areolar tissue.

7. A delicate areolar tissue connects the vagina to the bladder and to the rectum, except in the region of the hæmorrhoidal mesenteroid of the vagina.

8. The lateral ligament of the uterus is thinner and wider than the corresponding lateral ligament or mesenteroid of the vagina.

9. The lateral mesenteroid of the vagina is a trapezoid support formed by the vaginal portion of the uterine and the vaginal vessels as they traverse the fibro-areolar tissue lateral to the vagina.

10. The anteroposterior flattening of the uterus and the vagina is due to the lateral attachments.

11. The cylindrical shape of the cervix is due to the absence of a lateral attachment, and the preponderance of circular fibers.

12. The round and uterosacral ligaments are fibromuscular bundles in which the muscular tissue predominates.

13. Side-to-side closure of the vaginal vault following complete hysterectomy will increase the efficiency of the lateral attachments.

14. Plastic operations increase the general pelvic tone by increasing the tone of the lateral mesenteroid of the vagina.

15. The passage of the middle hæmorrhoidal vessels from the anterior surface of the rectum to the posterior wall of the vagina forms a distinct mesenteroid which divides the rectovaginal area into a proximal four-fifths and a lower one-fifth.

16. Postpartum cervical inspections are facilitated by traction which is placed laterally rather than anteriorly and posteriorly.

17. Sulcus tears are often serious due to a laceration of the vaginal plexus.

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THE RETROPERITONEAL SYNDROME AND THE RELATION BETWEEN KIDNEY AND GASTRO-INTESTINAL REFLEXES

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EVERY surgeon, at least once in his career, has had this experience: a patient comes to him with abdominal pain with vomiting, distention, arrest of fæces, and gas. The diagnosis of intestinal occlusion or peritonitis seems to be beyond question, although the cause of the ileus or peritonitis is not very evident. Emergency operation seems to be necessary and when performed shows no liquid in the abdomen, the intestine dilated in some places and in others contracted and filiform, the appendix, stomach, duodenum, and pelvic organs apparently normal. Operation is performed and often the patient recovers, as if the operation had put a stop to the spasm or paralysis of the intestine.

We believe that some of these intestinal occlusions which are seemingly without cause, may be due to disease of the kidneys or to a pathological irritation of the posterior parietal peritoneum. One of us in 1897 noticed that an irritation of the parietal peritoneum may cause a series of reflexes which produce what is called "abdominal shock" or "peritonism." It, therefore, seemed probable to us that reflexes of the same kind might originate in the kidney or perirenal region.

The observation of numerous clinical cases confirmed this hypothesis. For some time past it has been known that a nephritic colic may manifest itself by abdominal and peritoneal phenomena intense enough to suggest ileus (Sternberg, Stewart, Quénu). In the literature several cases are reported in which the patients were operated upon for a suspected occlusion but operation did not show the cause of the distress. Some hours later, however, these patients passed small ureteral calculi and at the same time had bowel movements.

We have also noticed in some patients with hydronephrosis that the condition was manifested only by an acute abdominal syndrome without urinary symptoms. We reported some such cases in 1909 in Maire's thesis. Several authors have since discussed this subject, Samuels, Kern, and McGlannon having reported cases of hydronephrosis with gastro-intestinal symptoms.

One of us has collected a number of cases not only of kidney lithiasis and hydronephrosis but also of other surgical diseases of the kidney, such as tuberculosis, tumor, polycystic kidney, and pyonephrosis in which the conditions were manifested only by an abdominal syndrome with no urinary symptoms whatever.

Certain perirenal affections may also cause an acute abdominal syndrome. Extraperitoneal rupture of a hydronephrosis (von Saar), spontaneous perirenal hæmorrhage (Grasman, Greco, etc.), and beginning perinephritic phlegmon (Tixier) often resemble intestinal occlusion or peritonitis. Frequently patients with these conditions have been operated upon on a diagnosis of acute appendicitis.

Following traumatism, an effusion of blood under the posterior parietal peritoneum (retroperitoneal effusion) sometimes causes a syndrome simulating peritonitis or intestinal occlusion: the following is a résumé of a characteristic case of that kind (Tixier).

A woman, aged 28 years, was in an automobile accident on December 12, 1927, at half past nine o'clock. Seen some hours later she showed signs of violent dorsolumbar contusion. Her abdomen was distended; she vomited twice. There was no sign of fracture of the spinal column or of the pelvis. Roentgenograms of the pelvis and of the lumbosacral spinal column were negative. The absence of lumbo-abdominal contracture and the pulse of 80 argued against the hypothesis of rupture of an abdominal organ. The next day the abdominal symptoms were worse; the patient was agitated, vomited bile, her pulse rose to 100; there was intense meteorism. A median laparotomy was made. The liver

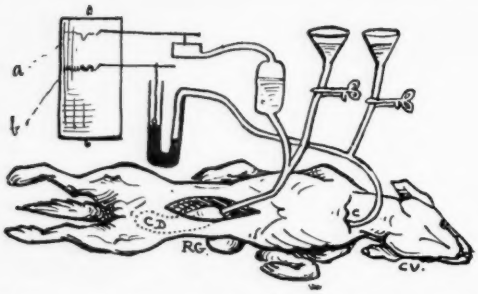

Fig. 1. Graphic registration of intestinal contractions and arterial pressure during stimulation of the kidney. *CD*, Exploratory capsule placed in the intestine (tracing *a:* intestinal contractions). *RG*, Lombotomy for stimulation of the left kidney. *C*, Registration carotid pressure (*b*).

and spleen did not show rupture; the mesenteric artery was intact; there was striking distention of some loops of intestine in contrast with the collapse of other parts of the small and large intestine. The abdomen was emptied and examined thoroughly. There were no tears and no perforations. The manipulation of the loops of intestine brought about a copious evacuation on the operating table with passage of a large amount of intestinal gas. As a result of the deflation of the intestines a large suffusion of black blood was found under the posterior peritoneum which descended along the left side of the spinal column and extended into the root of the mesentery. A retroperitoneal hæmatoma of unknown origin was diagnosed. The hæmorrhage seemed to be arrested. The meteorism and distention of segments of the intestine had produced a retroperitoneal syndrome. There was no hæmaturia. The operation was well borne; some gas was passed the next day, but the signs of occlusion soon began again, and the patient died on December 17. Autopsy was not allowed.

PATHOGENESIS

From this instance it seems probable that an irritation of the kidney by a calculus or by a hydronephrosis or the sudden stretching of the posterior parietal peritoneum by an effusion of blood, urine, or pus may cause a contracture or paralysis of the intestine which clinically simulates an ileus or peritonitis without there being any intra-abdominal or intestinal lesion. How is this to be explained? It seems logical to suppose that the condition may be caused by motor or inhibitory reflexes of the intestine, the point of origin of which is in the sensory nerves of the kidney, the ureter, or the posterior parietal peritoneum.

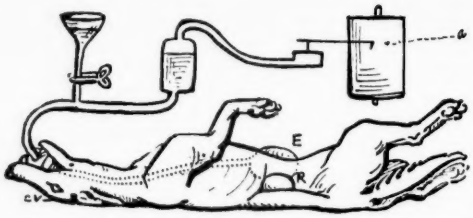

Fig. 2. Graphic registration of stomach contractions during stimulation of the kidney. *E*, Exploratory capsule introduced into the stomach by means of an œsophageal sound (tracing *a:* gastric contractions). *R*, Lombotomy for stimulation of the kidney.

As a matter of fact it has been shown by dissection that many anastomoses exist between the renal plexus and the superior and inferior mesenteric plexuses. We have, therefore, tried to show the presence of reflexes from the kidney to the digestive tract which follow the route of these anastomoses.

1. Effect of retroperitoneal injections. Guinea pigs were used for these first experiments. Rapid injections of physiological salt solution at 30 degrees were made behind the posterior parietal peritoneum. In a few minutes the peritoneal cavity was opened through a large incision and the intestine was carefully examined throughout its length.

In several guinea pigs special changes were found: some segments were very much dilated and congested, as if affected with vasodilatation and paralysis; the intermediate segments were pale, contracted, and filiform. In a control guinea pig that had not been given an injection only the peristaltic movements of the intestine as it came in contact with the air were noted (the animal should not be anæsthetized or handled too roughly, for asphyxia causes peristaltic movements). We believe that there is a relation between the renal and digestive reflexes in patients with the retroperitoneal syndrome.

2. Influence of renal and perirenal stimuli on gastro-intestinal motility. In order to verify the influence on the movements of the stomach and intestine, of stimuli of the kidney, of the mucous membrane of the pelvis and of the prerenal peritoneum, experiments were carried out on dogs. Tracings of the contractility of the digestive tract were registered by means of an exploratory ampoule filled with warm

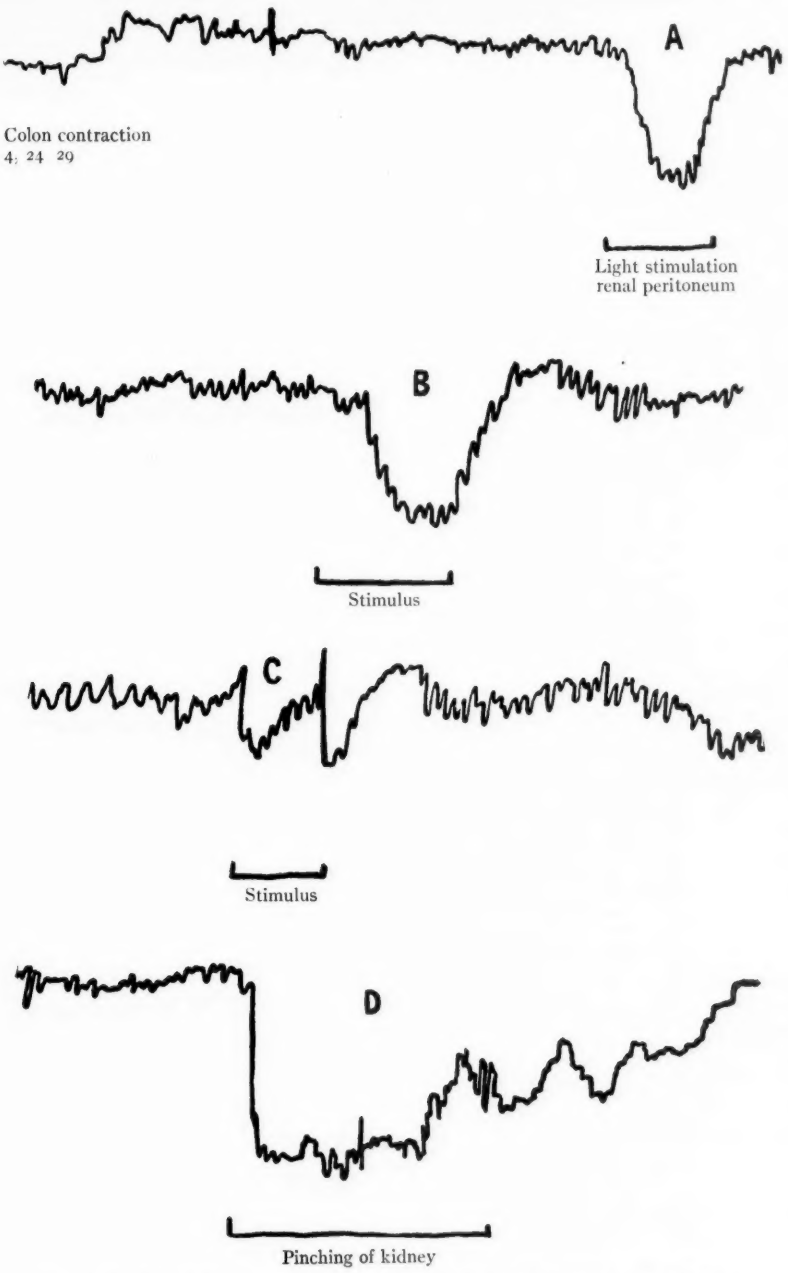

Fig. 3. Tracing of the experiment shown in Figure 1. Each stimulus of the kidney region caused an inhibition of contractions of the colon.

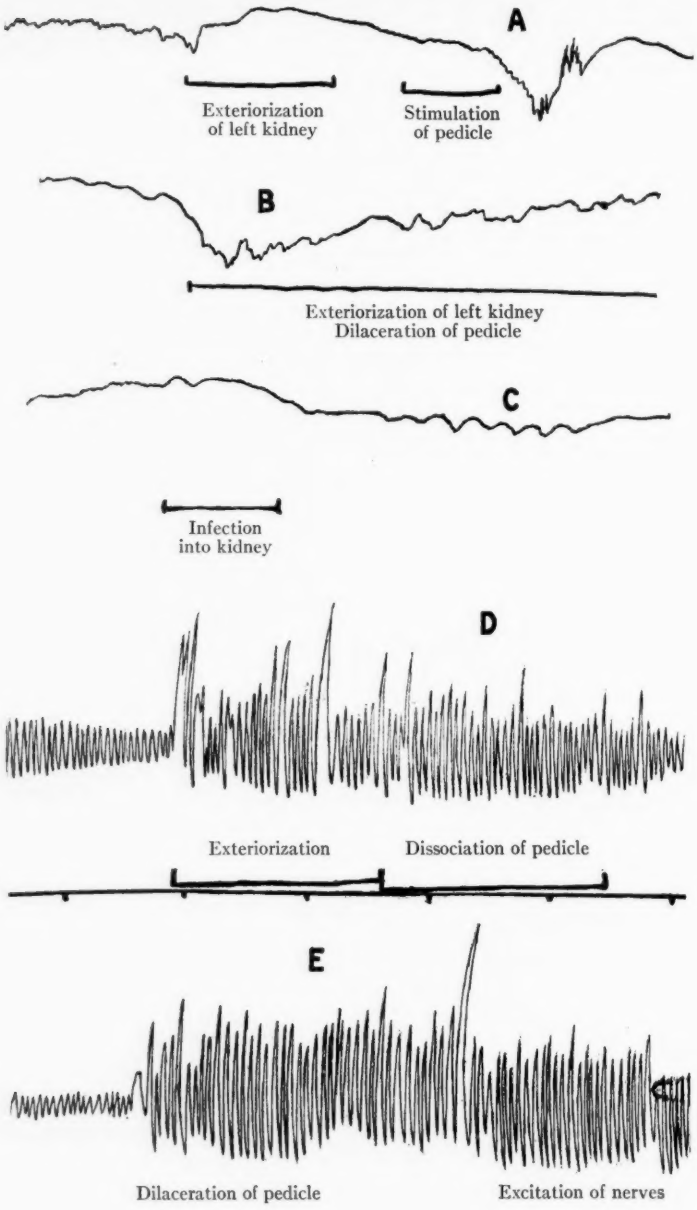

Fig. 4. *ABC*, Another tracing of the experiment in Figure 1. Series of peristaltic waves of the colon obtained by stimulation of the kidney. *DE*, Tracing of the experiment of Figure 2; registration of gastric contractions in the course of renal stimuli.

water placed in the stomach or intestine and connected with a Marey's kymograph (Figs. 1 and 2). The tracings show the effect of renal stimuli on the motility of the intestine (Figs. 3 and 4).

From the results of these experiments and from clinical observations we have been able to collect, we believe that we are safe in saying that there are kidney reflexes which affect the gastro-intestinal tract. These reflexes may explain the curious abdominal syndromes sometimes found in the presence of kidney disease, and would explain the pathogenesis of what we have called the retroperitoneal syndrome.

PHYSIO-PATHOLOGICAL CONCEPTION OF THE RENO-DIGESTIVE REFLEXES

In the light of these anatomical and experimental data, theories may be evolved which may be found useful both in clinical and operative ways. Let us take up successively the point of origin, and the center and centrifugal routes of these reflexes.

Point of origin of the reflexes. Stimulation of the pyelo-ureteral mucous membrane may be caused by a calculus, a blood clot, or a lump of pus.

Stimulation of the pelvic nerve terminals by distention is brought about by congenital or calculous hydronephrosis, etc. and occurs in the course of ureteral reflexes.

In the presence of tuberculosis and tumors of the kidney, stimulation of the kidney substance by a pus focus or hæmorrhage under tension, is seen. Reflex phenomena are not produced unless lesions of this kind are juxtacortical.

In operations for floating kidney, particularly in nephropexy when the kidney is not entirely freed from adhesions, traction is brought about on the fibers of the renal plexus and indirectly on the solar plexus. A perirenal hæmorrhage causes distention and laceration of these nerve fibers or the solar plexus itself may be affected.

Traction on the prerenal peritoneum or its direct stimulation may be produced by perinephritic phlegmons or hæmatomata or by a hæmorrhage which suddenly distends a tumor or a polycystic kidney.

Centers and points of reflection. The solar plexus seems to be the usual center of reflection. The phenomena seen clinically are very much like the syndromes of solar stimulation and paralysis described in 1853 by Claude Bernard and more recently studied by Laignel-Lavastine, Glockner, Exner and Jaeger, and others.

There are perhaps reflexes with a shorter circuit which pass through the renal ganglia and the inferior mesenteric ganglion; but there are certainly reflexes which have a longer circuit arising in different segments of the cord. According to Kappis and Gino Pieri, the twelfth dorsal and first dorsal lumbar segments correspond to the kidneys.

An important question rises here: the different irritations discussed are produced very frequently but the centers do not always respond to them. Reno-digestive reflexes are not very frequently seen. It seems to us that several conditions are necessary to make these reflexes evident:

1. Necessity for summation of the irritations. Experimentally an isolated renal stimulus does not always cause a response on the part of the digestive organs. More constant, more intense, and more durable effects are obtained by the summation of stimuli of different natures. For instance, when cortical stimulation of the kidney has failed, a combination of this stimulus with slight traction on the pedicle of the kidney or a distention of the pelvis and ureter is often effective.

Clinically the same thing is true, and the renal affections which bring about digestive reflexes always do so by a complex mechanism. Lithiasis is always associated with pelvic distention and traumatism of the mucous membrane of the pelvis by the calculus; hydronephrosis is associated with distention of the pelvis and traction on the prerenal peritoneum; in perirenal hæmorrhage the direct stimulus of the nerve plexus is associated with distention of the peritoneum by the blood effusion.

2. Individual predisposition. It is true that certain individuals are predisposed to reno-digestive reflexes. While this predisposition has been attributed to vagosympathetic disequilibrium, this explanation is as yet only

a hypothesis. However, in several patients, recurrent accidents of different kinds have been noted: for example, in patients who have had nephritic colic associated with intestinal occlusion reflex ileus occurred after an operation on the kidney.

3. Sensitization of the nerve centers by an infection or intoxication. Experimentally an injection of pilocarpin greatly increases the intensity of the kidney reflexes to gastro-intestinal tract and lowers the threshold of stimulation necessary to bring them about. Atropin and nicotine generally have the opposite effect. It is permissible to believe that a pre-existing intoxication or infection acting on the solar plexus may change its reflex capacity and produce reflexes which without that would not have become manifest. We believe that uræmia, even if slight, frequently plays this part of sensitizer; attacks of transitory uræmia in the course of kidney disease and after operation are now well known; they may produce an intense effect on the nervous system. These hypotheses require further research.

Centrifugal routes of kidney reflexes to gastro-intestinal tract. These reflexes may reach the cerebrospinal nervous system and produce pain which is localized by the patient in the skin zone which is the projection of these nerves (referred pain). Head carefully studied this pain projection on the wall of the abdomen in the course of the attacks of pain in kidney disease and established both their topography and their probable mechanism. This pain is associated with muscle contraction and may simulate an acute abdominal disturbance; that is why we consider it in connection with renodigestive reflexes.

But the centrifugal route of the true reno-gastro-intestinal reflexes is the vegetative nerves—the pneumogastric and particularly the sympathetic. The fact that these nerves are affected is easily verified in the presence of nephritic colic: for example, there are changes in the pulse, blood pressure, vasomotor disturbances, etc. (Pal, Potain, F. Franck, Paillard).

From the point of view of their gastro-intestinal effects these renal reflexes may be classified according to the organ in which they predominate:

1. Reno-gastric reflexes. They are motor, secretory, and vasomotor. Sometimes they are phenomena of stimulation, sometimes of inhibition. The pyloric sphincter is particularly sensitive to these stimuli. Carnot, Satre among others have verified this experimentally.

2. Reno-intestinal reflexes. Some reflexes are secretory and vasomotor, others motor. Sometimes spasm is produced, sometimes paralysis. The large intestine is particularly sensitive to these reflexes; the rectum may be the site of similar phenomena, particularly in the course of violent ureteral irritation (Loeper).

3. Peritonism of renal origin. These different reno-intestinal reflexes may be associated with each other or combined with cardiac, respiratory, or vasomotor reflexes to cause a group of symptoms resembling those of peritonitis. The clinical picture is then made up of pain, contracture of the wall, distention, vomiting, arrest of fæces and gas, and rapid pulse without fever.

This, in our opinion, is the pathogenesis of the retroperitoneal syndrome.

For the bibliography the reader is referred to the thesis of Charles Clavel: Peritoneal and Gastro-intestinal Syndromes in Surgical Diseases of the Kidney. Lyon: Camugli, 1929, p. 326, fig. 16.

PATHOLOGICAL STUDIES ON INJECTED VARICOSE VEINS[1]

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THE injection treatment of varicose veins of the extremities is based upon the theory that the injected substance injures the vascular endothelium and causes the formation of a thrombus at the site of injury. The thrombus is believed to close the lumen of the vessel completely and when it becomes organized the vessel is entirely obliterated. In spite of a rather extensive literature upon the results of this method of treating varices, considerable uncertainty exists as to the exact nature and extent of the processes involved. The theory as usually stated does not explain the wide variations in the extent to which a vessel may be occluded by a single injection of a relatively mild solution. It does not explain the difference in the consistence of the occluding mass in various portions of the same injected veins, nor does it explain the partially successful treatments and the recurrences. The present investigation was undertaken with the hope that careful microscopic examination of treated veins might throw some light upon these rather perplexing questions.

Relatively few investigators have studied microscopically the changes that take place in artificially thrombosed varicose veins. Bazelis found that 24 hours after the injection of 1 cubic centimeter of mercury biniodide, the endothelium was swollen and partially separated from the vessel wall. No clot or thrombus was present; no leucocytic infiltration had occurred. At 48 hours, proliferation of endothelial cells had occurred to such an extent that the lining membrane was unrecognizable. Fibrin had been deposited on the vessel wall. At 72 hours a clot had entirely obliterated the lumen. Regard studied the veins of dogs and rabbits 1 to 4 days after the injection of irritating fluids. He found that the vessel wall became œdematous. After the first day the endothelium was detached from the vessel wall. Fibrin then collected on the wall, and after the third day the lumen was obliterated by a thrombus. Sicard and Gaugier stated that in the first stages the endothelium became hypertrophied. In the second stage fibrin collected over the whole injured area. Upon this basis a clot formed firmly adherent to the vessel wall.

Meisen found that 2 hours after the injection of a 40 per cent sodium salicylate solution into the median vein of a horse, the endothelium was greyish for a distance of 6 inches from the point of injection. Microscopically, red corpuscles were adherent to an apparently normal endothelium, occasionally in large masses, but mostly as a continuous lining, gradually becoming palisade rows of erythrocytes. Twenty-four hours after the injection of a 30 per cent solution, a thrombus was formed so firmly adherent that force was needed to remove it with a knife. Microscopically, the large thrombus was nearly filling the lumen, and so closely attached to the vessel wall that no intima was to be seen. The adventitia showed an intense inflammatory reaction. With sodium citrate similar results were obtained, but the intravascular phenomena were less marked while the extravascular changes were more marked. Organization was noted on the sixth day.

Doerffel studied the ear veins of rabbits after injections of 30 per cent sodium chloride and 50 per cent grape sugar. He came to the conclusion that the successful action of these substances rested upon their stimulation of the intima into an extremely vigorous state of proliferation, within and upon the resulting meshwork of which a coagulation thrombus developed. Wolf injected the ear veins of rabbits with 1 per cent solutions of bichloride of mercury. He found that in the early stages the thrombus filling the lumen was clearly attached to the vessel wall at a point where the intima had become detached. The intima was found embedded in the thrombus, thus guaranteeing firm fixation of the mass. The loosening of the intima did not occur throughout the whole vein, but only at separate points. At 2 days the above process had so

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extended that the whole intima was necrotic. No endothelium could be seen, but at the margin of the lumen were large numbers of nuclear fragments. There were numerous clefts and depressions in the inner portion of the vessel wall, and thrombus filled these spaces, thus further immobilizing the occluding mass. At 5 to 7 days, leucocytic infiltrations of the vessel wall were noted, and increased numbers of polymorphonuclear leucocytes and lymphocytes were found in the thrombus. In the eighth and ninth day specimens, organization was beginning. After a month, organization was complete.

Schwarz and Ratschow found that 24 hours after the injection of a 50 or 60 per cent solution of calorose into the ear vein of a rabbit disintegration of the intima began, and that a fine network of fibrin containing erythrocytes filled the lumen. Following this, necrosis of the endothelial cells progressed and the fibrin network became denser. Evidences of exudative inflammation were seen in the vessel wall, and these changes reached their maximum on the fourth day, at which time a well stratified thrombus had developed. In 3 to 4 weeks, the vessel could scarcely be recognized.

Binet and Verne studied the ear veins of rabbits after injection of 30 per cent solutions of sodium salicylate and found that the endothelial cells became hypertrophic, assuming the form of undifferentiated mesenchyme. These elements then took part in organizing clot.

Kern and Angle caused thrombosis of the jugular vein in dogs by injection of 20 to 30 per cent sodium chloride solution in small doses (0.12 to 0.5 cubic centimeter). The specimens removed at 48 hours showed a very firm thrombus that was not easily dislodged. The vein wall was infiltrated by leucocytes, and no endothelial cells were seen. After 1 week a firm thrombus occluded the distal segment. The thrombus was half red and half white, and tapering. Plasma cells were beginning to grow into the thrombus. After 2 weeks the thrombus was about two-thirds organized. In none of the 28 dogs used for the experiments did evidence of embolism or infarction of the lungs develop.

Howard, Jackson, and Mahon have recently described the histological findings in a larger series of injected veins than had hitherto been reported. Thirteen separate veins were examined from 24 hours to 10 months after injection. Proliferation of endothelial cells into the thrombus was found at 72 hours, and organization was distinct at 9 days. Complete organization and fibrosis had occurred at 9 months.

It is to be noted that in most of these observations the words clot and thrombus are used synonymously. A deposition thrombus is a solid mass formed in a vessel through which blood is flowing and is composed of the solid elements of the blood, with or without fibrin. A moving stream of blood is essential to its formation. Coagulation thrombus, however, is formed from stagnant blood, and may occur at any time that the conditions necessary to produce coagulation are present. It is nothing more than an antemortem clot. It is composed almost entirely of erythrocytes held together by an interlacing network of delicate strands of fibrin. Platelets are very inconspicuous and never occur in large masses. Leucocytes are present in no greater numbers than in the circulating blood, and occur singly, well distributed among the erythrocytes. The deposition thrombus has a characteristic microscopical appearance, due to its manner of development. All deposition thrombi contain the separate solid elements of the blood in more or less distinct aggregations. Closely packed together are laminations composed of red cell masses, platelet masses, leucocytes, and fibrin. These elements may vary considerably in the proportion they occupy of the whole. If leucocytes, platelets, or fibrin predominate, the structure is a white thrombus. If erythrocytes predominate, the mass is called a red thrombus. Factors predisposing to thrombus formation are slowing of the blood stream, increased coagulability of the blood, and roughening of the intima of the vessel. Since, in a varicose vein, the blood flows very slowly, thrombosis should be obtained easily by injuring the vascular endothelium.

METHOD OF STUDY

The early changes produced in varicose veins by the injection treatment were studied

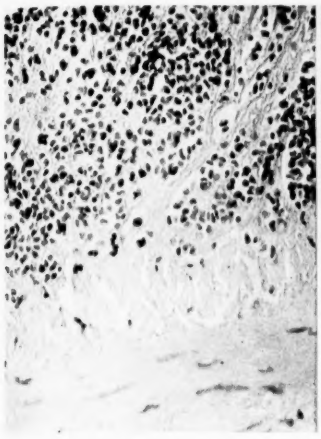


Fig. 1.

Fig. 2.

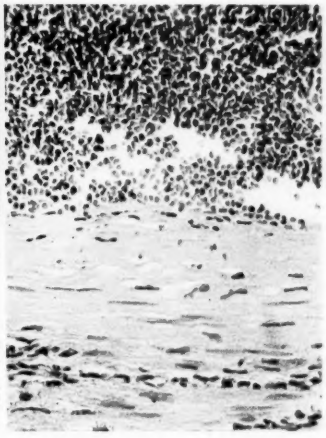


Fig. 3.

Fig. 1. Severe intimal injury underlying laminated thrombus. Twenty-four hours after the injection of urethane.

Fig. 2. Severe intimal injury underlying simple clot. Twenty-four hours after injection of a 75 per cent solution of invert sugar.

Fig. 3. Loose clot overlying uninjured intima at a point 4 centimeters from the section shown in Figure 2.

by the examination of segments from the varicose saphenous veins of six patients presenting themselves for treatment at the varicose vein clinic of the Minneapolis General Hospital. The specimens were removed 24 to 48 hours after the injection of urethane, or strong sugar or salt solutions. At the time of removal all of these were filled by a solid mass which varied somewhat in firmness in different portions of the single specimens. Immediately after removal accurate outline drawings were made to the exact size, and the specimens were fixed in 10 per cent formalin. With a sharp razor, 1 millimeter sections were taken at intervals of 2 to 4 millimeters through the whole length of the specimen. These blocks were numbered serially, sectioned, mounted, stained with hæmatoxylin and eosin, and studied serially.

Changes in the vessel wall. Evidences of acute inflammation were found in all of these vessels. The adventitia invariably contained scattered infiltrations of polymorphonuclear and mononuclear leucocytes. In none was necrosis noted, and in none were the accumulations so dense as to suggest abscess formation. Rarely were more than 50 or 60 leucocytes seen under one high power field. The intensity of this reaction varied considerably in various portions of the same vessel. In one section the changes might be marked, while a centimeter or two distant, only an occasional leucocyte could be found. The reaction in the media was exactly similar. The leucocytes here were found scattered between the muscle fibers and much elongated in shape. Here also there was no reaction of such severity as to suggest even early abscess formation. As in the adventitia, there were frequently segments in which the media was almost free from leucocytes, while an adjacent or neighboring section showed fairly dense infiltration. Where the reaction was most intense, the muscle fibers frequently seemed stretched apart, as if by œdema. In general, these inflammatory changes were most severe in the region of the vessel filled by deposition thrombus, and were unusual in those portions where clot filled the lumen. However, occasionally the most intense reaction was in a segment filled only by clot, where the intima was still intact. Likewise, the changes in the endothelium differed considerably in different portions of the same vessel. In each specimen the following variations in the state of the endothelium were noted. (1) In some portions where the deposition thrombus was adherent the endothelium was unrecognizable (Fig. 1).

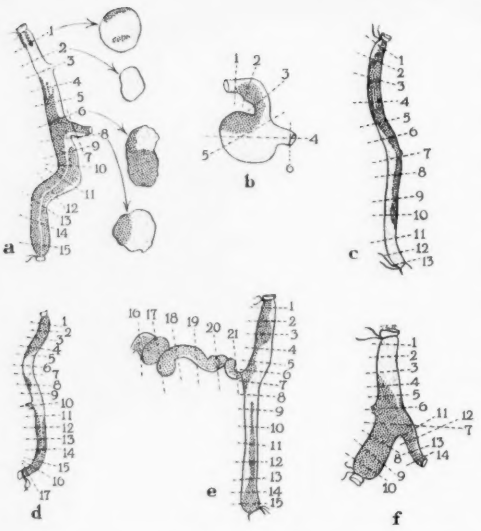


Fig. 4. Diagrams showing the structure of the occluding mass in 6 specimens removed 24 or 48 hours after injection of urethane, calorose, or sodium chloride solution. The fine stippling represents thrombus; the coarse stippling represents clot. The numbered dotted lines represent levels at which blocks were removed for microscopic study. *a* includes the appearance of the vessel in cross section at a number of typical levels.

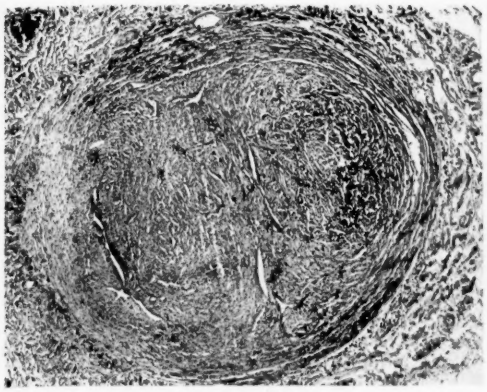
(2) Occasionally if there was only clot the endothelium was destroyed (Fig. 2). (3) In other portions normal endothelium was found to underlie true thrombus. It would seem that to develop a deposition thrombus the endothelium must be injured, but that thrombus does not necessarily develop upon all injured endothelium. It seems further that thrombus may at least extend itself over areas of normal endothelium.

Gross structure of the thrombus. The observations on the occluding mass were largely to determine whether the material consisted of clot or of deposition thrombus. By shading, in the outline drawing of the vein, the portions found in the cross sections to contain deposition thrombus, a fairly accurate representation is obtained of the whole mass which fills the vessel (Fig. 4). These drawings necessarily do not represent the structure of the mass in detail, since depth cannot be shown; yet we are convinced that in all essentials the mass is as represented. In the absence of true laminations, as in the terminal portions of the thrombus, masses of fibrin were considered as representing deposition thrombus. It has been observed repeatedly that the fibrin of a rapidly formed clot occurs in irregular filaments and delicate strands that form a mesh or network in which the erythrocytes are entangled.

As may be seen in the accompanying diagrams, each vein had been occluded in exactly the same manner. The lumen in each specimen was completely filled by a solid mass of blood; but of this mass, only a portion consisted of deposition thrombus. The remainder was formed of simple blood clot. The deposition thrombus represented about one-third of the occluding mass, and only a very small portion of this completely filled the lumen. This finding is in entire accord with the theory of thrombus formation and with the modern coagulation theory.

MECHANISM OF OCCLUSION

It is well known that injured tissues and platelets liberate a substance, tissue extract (Mills' tissue fibrinogen), which is capable of precipitating fibrin from normal blood plasma. It is equally well known that in the formation of fibrin a substance (thrombin) is elaborated which is able to induce coagulation of normal plasma. Occlusion of the injected varicose vein begins with injury of the intima by the injected substance. Upon the injured area platelets are deposited and a deposition thrombus develops. As soon as the thrombus fills the lumen its growth ceases. At the junction of the thrombus and the now stagnant uncoagulated blood there will be present coagulating substances (tissue extract from the platelets in the thrombus) which will at once precipitate fibrin. This will cause an extension of the clot by the liberation of thrombin as fibrogen is changed to fibrin. The extent of this clot or coagulation thrombus will be determined by the number and size of the tributaries and the rapidity of circulation through them. If tributary veins are numerous and their circulation active, only a short segment of the vessel will be occluded. If the opposite conditions exist occlusion may be very extensive. Because of dilution of the



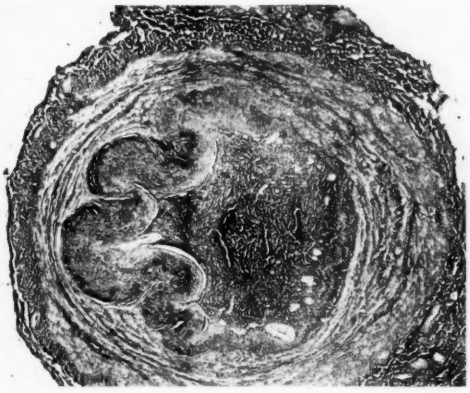


Fig. 5, left. Segment of a vein 605 days after artificial thrombosis. Fibrous tissue completely obliterates the lumen. The small spaces in the central portions represent dilated capillaries, and are probably remnants of those which took part in the process of organization. Note the preservation of the media.

Fig. 7. Specimen removed 760 days after injection. In this portion of the vessel obliteration has probably taken place by two separate processes. At the right the lumen is filled by hyalinized fibrous tissue containing traces of blood pigment. At the left there has been a tremendous hyperplasia of the intima, probably into an empty "cleft" or an area of softened blood clot. The thickened intima contains no blood pigment.

clotting substances, a clot will probably never extend into the deep veins of the leg or beyond the saphenofemoral junction so long as the circulation in these vessels is competent.

The essential facts concerning the development and structure of thrombi have been known for years, but they seem to have been overlooked by writers on the injection treatment of varicose veins. As early as 1899 Welch stated that the first stage is a white or accumulation thrombus, while the completed thrombus is a coagulum. In venous thromboses he noted that after the white thrombus has become occluding, the column of blood to the nearest branch or confluence is brought to a standstill and forms a red (coagulation) thrombus. Aschoff speaks of the primary, laminated portion as a deposition thrombus and states that this blends into and becomes continuous with the more extensive red portion or coagulation thrombus.

ORGANIZATION

The progress of organization was followed by the examination of segments of varicose veins removed from 44 additional patients treated in the varicose vein clinic of the Minneapolis General Hospital. The segments were removed 4 days to 760 days after injection. The specimens used for this purpose were all obtained from the medial aspect of the thigh a few centimeters above the knee, and special effort was made to remove the segments only from veins which were approximately 1 centimeter in diameter at the time treatment was given. In these vessels, organization was found to begin with penetrations of fibroblasts and capillaries into the thrombus on the fifth day after injection. On the forty-seventh day these proliferating

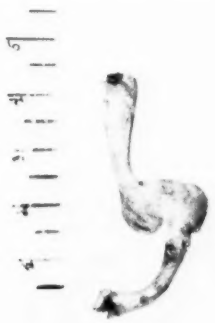
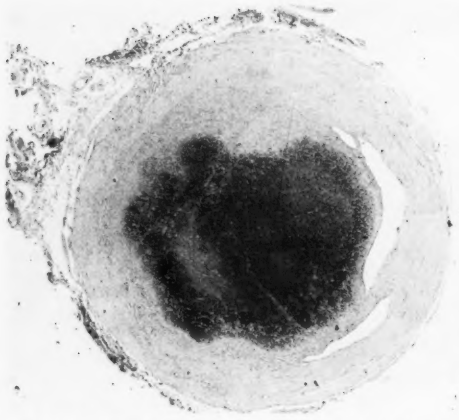


Fig. 6. Segment of vein removed 760 days after injection. The narrower portions were completely occluded by hyalinized fibrous tissue. The dilated portion contained a semifluid chocolate colored mass of hæmolized blood.



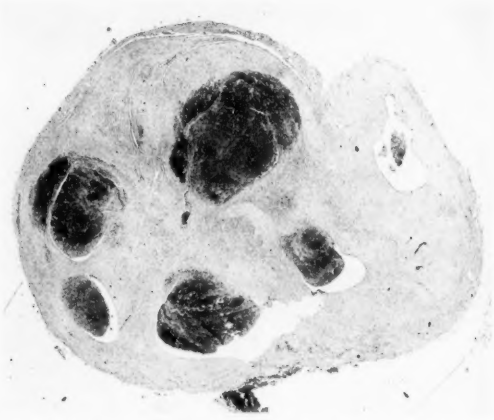


Fig. 8, left. Section of a vessel 40 days after injection. The central mass is of clotted blood, through most portions of which is an abundant growth of fibroblasts. At the side are two large clefts lined with endothelium. These probably result from retraction of the clot before organization has begun.

Fig. 9. Section of vein from a patient who returned with clinical recurrence 218 days after treatment. The vein was again injected, and the specimen was removed 12 days later. The central portions are formed of dense fibrous tissue containing blood pigment. At the periphery are a number of large sinuses. Some of these contain clot and others thrombus as a result of injections 12 days previous to removal.

tissues had penetrated to all portions of the thrombus. From this time on there was progressive increase in collagenous fibers with absorption of the elements of the thrombus, until 605 days later the space once occupied by thrombus had become a mass of extremely dense hyalinized fibrous tissue containing only traces of blood pigment. In the fibrous tissue were occasionally small dilated capillary spaces containing normal blood cells (Fig. 5).

Organization advanced at a fairly even rate, so that after some experience one could usually estimate to within the limits of a few days the age of the thrombus or clot. The speed and completeness of the reaction seemed to have no correlation with the thickness of the vessel wall or the state of its nourishment, or with the presence or absence of inflammatory changes in the adventitia. However, the progress was not absolutely uniform, as occasionally organization had progressed less in an older thrombus than in a younger. Thus the process was more complete in the eighty-ninth day specimen than in that 109 days old. It was often noted, especially in specimens more than 100 days old, that in certain portions of the vessels organization seemed to have come to a standstill, with dense fibrous tissue containing pigment at the periphery of the vessel lumen, and semifluid hæmolized blood remaining in the center. These areas in the gross specimens, though greatly reduced in caliber, were still considerably dilated beyond the diameters of other portions of the vessel, and tended to give the vessel segment a beaded appearance (Fig. 6). It seems probable that at the dilated portions the clot of blood was too soft to support the ingrowth of new tissue. Occasionally segments of vessels were found closed by the mechanism of intimal thickening. In these the lumen had become simply an irregular slit, lined by endothelium and surrounded by a tremendously thickened and fibrous intima free from blood pigment. The appearances were exactly similar to those in disuse atrophy of a vessel, and probably represent the fate of segments of the injected vessel in which the clot has undergone lysis and absorption without organization (Fig. 7). The muscular coats even in the latest stages were surprisingly well preserved. Although all of the sections showed minor changes in the smooth muscle fibers, such as hydropic degeneration and hyalinization, and in a number of specimens there was an apparent increase of fibrous tissue between the layers of

smooth muscle, no changes were found which could be ascribed with certainty to the injections. Even in the vessel removed 763 days after treatment, the identity of the media was clearly maintained (Figs. 5 and 6). These findings contradict those of Bazelis, who described almost complete destruction of muscular elements at 3 months.

Several different solutions were used to cause thrombosis in the veins studied. These were calorose (75 per cent invert sugar with 5 per cent saccharose); quinine sulphate, 12 per cent with urethane, 6 per cent; sodium chloride, 20 per cent; invert sugar, 35 per cent with sodium chloride, 15 per cent; sodium morrhuate, 10 per cent. There were no appreciable differences in the histological appearances caused by these different solutions. Following the injection of sodium morrhuate the inflammatory reaction seemed somewhat more intense than that caused by the other solutions, and the rate of organization seemed slightly more rapid. However, too few specimens were studied after the use of sodium morrhuate to warrant definite conclusions.

MECHANISM OF RECURRENCE

In a number of specimens cleft like spaces were found between the intima and the blood mass filling the lumen. These usually occurred at levels which were occupied by clot, and in the later stages they were completely lined with endothelium. They frequently contained normal erythrocytes, indicating circulation of blood through them (Fig. 8).

It has already been shown that simple clot forms the larger portion of the occluding mass in the treated vein. Inasmuch as retraction is a normal property of clot, it is probable that cleft formation results from retraction of the clotted portion of the occluding mass from portions of the intima insufficiently injured to cause its adherence. If retraction is so extensive that the resulting space between clot and vessel wall communicates between two tributary vessels opening into the principal lumen, the space at once becomes a blood filled sinus, and the vessel is again a part of the active circulatory system, with the potentiality of undergoing still further varicose dilatation. DeTakats and Quint have made note of similar findings in veins removed after injections, and they point out the possibility of this being an underlying factor in the development of recurrences in the treated varicose vein. Howard, Jackson, and Mahon report the presence of such clefts in a large number of their specimens. They re-emphasize the importance of these spaces in producing recurrences of varicosities. Three of our specimens were from vessels which had developed recurrences after treatment. The lumen of each was found largely filled by fibrous tissue containing blood pigment. At the periphery were greatly dilated spaces containing fresh blood. Although greatly distended and rounded, the position of these sinuses was exactly similar to that of the clefts described (Fig. 9). In none of these three specimens could the recurrence be ascribed to excessive dilatation of capillaries which had taken part in the process of organization.

CONCLUSIONS

Thrombus formation in varicose veins treated by injection of even "mild" solutions, depends upon an injury to the endothelial lining of the vessel. Deposition thrombus usually forms the smaller portion of the total occluding mass developed after treatment. The larger mass consists of simple clot.

Recurrence probably results from retraction of the clotted portion of the mass from the vessel wall between tributaries of the main channel, with re-establishment of circulation through the new channel.

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CLINICAL SURGERY

FROM THE TORONTO GENERAL HOSPITAL

THYROIDECTOMY

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THE pre-operative, the operative, and the postoperative treatment of goiter has undergone, during the past 10 years, changes which have been, more or less, revolutionary. The pre-operative changes in management have been directed toward placing the patient in a much more favorable position for operation. The operative improvements have been directed toward the avoidance of operative complications and postoperative recurrence. The postoperative regimen has been directed toward minimizing the postoperative reaction.

Enough time has elapsed so that our ideas have become crystallized, as is evidenced by the fact that there has been very little added by way of improvement during the past 3 or 4 years. We may, therefore, with profit review the important points in goiter surgery which will at least remain important until further research discloses the etiology of goiter.

The important points in the technique of thyroidectomy have been impressed on surgeons by the operative and postoperative difficulties, and it is by critical review of these difficulties that one arrives at a basis on which to judge the efficiency of any operative method. The important points in technique are: the exposure of the gland; the delivery of the gland, which may or may not be difficult, depending on its degree of friability or its position; hæmorrhage; recurrent nerve injury; and parathyroid injury.

In order to judge the efficiency of the technique about to be described, a careful review was made of some 350 cases, taken in sequence, the idea being to learn, if possible, the complications directly attributable to operative technique.

No one can be dogmatic regarding recurrent laryngeal nerve injury unless the cases are followed by postoperative laryngoscopic examination. However, from a practical standpoint I may state that if there has been any postoperative respiratory difficulty or alteration in phonation, the surgeon must consider that he has created recurrent nerve injury until it can be proved otherwise.

The following technique has proved very satisfactory from every standpoint.

Step 1.—Elevation of the skin flap. The incision must be placed so as to be symmetrical as to extent on each side of the median line and as to level. It should not be too long, and rarely should it be necessary to extend it beyond the middle of the sternomastoid on either side. It is not the length of the incision which determines the adequacy of the exposure. The incision must be placed neither too high nor too low. If placed too high, the scar, if noticeable, cannot be covered; if too low, in time it stretches and becomes unsightly. With the head extended, the midpoint of the incision should be about 1.5 inches above the episternal notch.

After the skin flap is elevated from the fascia, all bleeding points should be secured and ligated with fine catgut. The field of operation should be made perfectly dry, and skin towels, made preferably of gauze, should be applied as in any other operation.

Step 2.—Exposure of the thyroid gland. To the occasional operator, adequate exposure is of prime importance and this is secured in one of two ways: by making ample division vertically of the fascia between the strap muscles; or later, by cross division of the strap muscles. The fascia on the surface of the gland is best divided by Mayo scissors. One must be careful that the last layer of fascia is separated from the gland to make delivery of the lobe easy. From now on the great secret of success in thyroid surgery is gentleness in all manipulations, and it must be remembered that toxic patients, particularly, will not stand trauma.

Step 3.—Separation of the strap muscles from the gland surface. This is done by careful dissection of the fascia and proper placing of ribbon retractors. During this stage the operator must decide as to whether or not the exposure will be

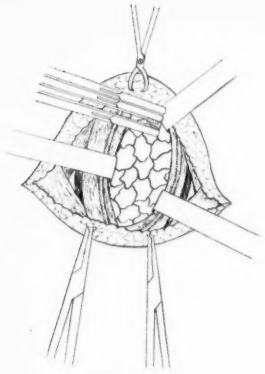

Fig. 1.

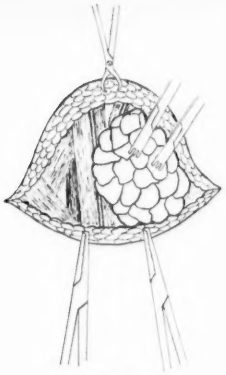

Fig. 2.

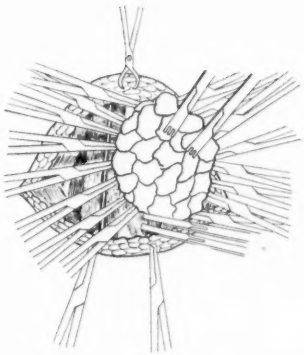

Fig. 3.

sufficient without dividing the strap muscles transversely. When in doubt always divide between forceps.

Step 4.—Clamping of the superior thyroid artery. As a rule, with blunt dissection the vessels readily come into view. They are triple clamped with Kocher forceps and divided between the distal pairs, double clamps being left on the proximal end. From now on Kocher forceps should not be used but smaller Crile forceps.

Step 5.—Delivery of the lobe. The upper pole should be delivered first and this is done by seizing the gland with Lahey forceps. A moderately enlarged lobe will generally roll out without difficulty, but, if there is a substernal projection, such is not the case. Never attempt to deliver the lower pole with the fingers unless all other methods fail. If the lower pole does not deliver readily, leave it alone until the upper pole has been liberated. This liberation of the upper pole allows the entire lobe of the gland to be dislocated upward and makes for easy delivery of the lower pole.

The recurrent nerve can be stretched or torn by digital delivery of the lower pole, and it would appear that this must be considered as one of the causes of recurrent nerve injury.

When the lobe is delivered, with gauze gently wipe backward the retroglandular fascia and grasp vessels with Crile forceps all around the lobe, being careful to keep forceps level and never to point them downward and inward and never to take large bites. Do not remove or pinch off any lobules of fat lest it be a parathyroid gland.

Step 6.—Amputation of the lobe. The aim should be to have a trough shaped piece of gland remain, but the amount to be left can be judged only by the type of case, the type of gland, and a great deal of experience in thyroid surgery.

The excision of the lobe is best done by cutting it on one side and then on the other, liberating some tissue at the upper pole and then some at the lower pole. Take time doing this and allow the assistant plenty of time to secure uncut vessels and bleeding points. At this stage, a reckless, panicky assistant can do a great deal of harm. He should hold a sponge in one hand and a pair of forceps in the other and follow along after the surgeon's knife, grasping bleeding vessels if he sees the bleeding point clearly. If he does not see the bleeding point, he should never guess at its location but should place a sponge of gauze over the area and then gradually sneak it off and get the bleeding point when it appears as the pressure is released. Blind grabbing of vessels by an assistant gives more surgeons "operative heart failure" than anything else. The assistant should also know the location of the trachea by feeling it with his fingers.

Step 7.—Ligation of the vessels. The superior pole should be doubly ligated and all other points singly, either with No. 1 or No. 2 catgut, but the latter size is best for the superior vessel. When all the vessels have been tied and all bleeding points stopped, the surface of the gland may be folded on itself by two or three interrupted sutures, the outer capsule being joined to the tracheal side. However, this procedure is not always necessary.

Step 8.—Closure. While the other lobe is being removed, the cavity left by removal of the first is packed with a teased out gauze fluff. When the second lobe has been removed the reconstruction work begins and the question of drainage is one to be settled. Drainage is rarely necessary with this technique, unless in the case of large substernal adenomata, but the advice again is, when in doubt, drain, but always be careful how you

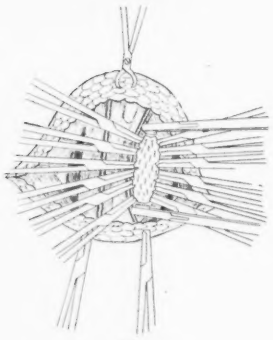

Fig. 4.

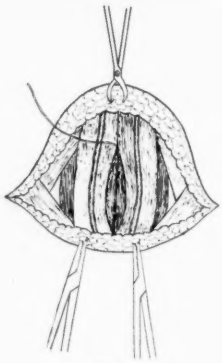

Fig. 5.

drain. Never drain through the midline but always laterally through a stab wound through the outer edges of the strap muscles. By staying away from the midline the troublesome "swallow-tug" is avoided, which, by the way, is not always such a simple thing to fix. If the muscles have been divided, they are sutured, if not, the sternohyoid muscle is sutured; approximating it by a continuous suture. The skin towels are removed and the skin closed with clips, no sutures being placed in the platysma. If drainage has been placed, it remains 36 hours, and if not, the wound needs no dressing until the clips are removed on the third day. As a rule, all dressings are off on the fourth or fifth day, and at the end of 10 days, some gentle massage is done by the patient to hasten the absorption of the operative exudate.

FROM THE LAHEY CLINIC

A FURTHER SUGGESTION FOR THE OPERATIVE TREATMENT OF PILONIDAL SINUSES

FRANK H. LAHEY, M.D., F.A.C.S., BOSTON

A PILONIDAL sinus, a developmental lesion, is a skin lined cyst located between the buttocks over the sacrum, frequently contains hair, and frequently becomes infected.

In a study of a series of 59 patients with pilonidal sinuses, we have found that, to insure against recurrence, it is necessary to remove the entire sinus tract, which means that wide bloc dissection of the entire sinus tract together with much of the subcutaneous fat surrounding it must be done. All extensions of the sinus, laterally and subcutaneously down to the fibers of the gluteus maximus on either side of the sacrum, must be dissected. In the middle line, the dissection must likewise be carried down through all of the subcutaneous fat to the aponeurosis covering the sacrum.

The dissection of a bloc of skin and subcutaneous tissue extensive enough to remove the entire sinus together with its ramifications and an often associated abscess, leaves a large defect over the sacrum, as is shown diagrammatically in Figure 2, b. The large defect thus produced fills in slowly with granulation tissue and when the wound is completely healed, there results a dense, thick scar directly over the sacrum which must of necessity be subjected to a great deal of pressure when the patient is sitting. This factor we have found to be the means of producing pressure necrosis of the scar. Therefore, healing can be accomplished only by relieving the area from pressure, but there is always the danger that the difficulty will return when the pressure is again applied.

To overcome this undesirable feature, we used a method which I described some time ago in this journal[1]. A pedunculated, fat lined skin flap was cut from one of the sides of the wound and was transferred to the midline, the free edge of the flap being sutured to the opposite edge of the wound (Fig. 3). In this way a fat lined skin pad was placed directly over the bony prominence of the sacrum. The lateral defect (Fig. 3), the result

[1] Surg., Gynec. & Obst. 1929, xlviii, 109-111.

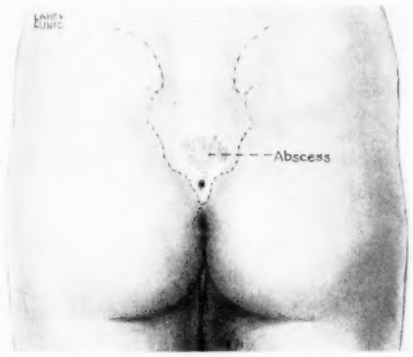

Fig. 1. Diagrammatic drawing showing the location of the opening of a pilonidal sinus. The shaded area indicates the extent of involvement by an abscess which may be associated with the sinus. The necessity for wide bloc dissection of these tracts to avoid leaving portions of the tract and to prevent recurrence is evident.

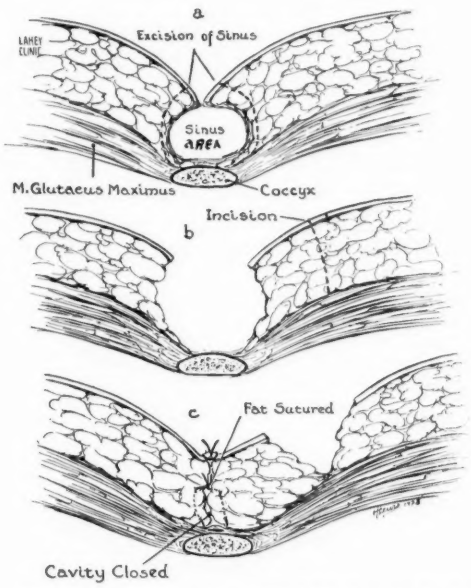

Fig. 2. This drawing is again published to emphasize *a*, the plan of bloc removal of the entire pilonidal tract; *b*, the large defect extending down to the sacrum; and *c*, how the complete separation of the flap (skin and subcutaneous fat) and its transference to the opposite side provides a fat lined flap directly over the bony sacrum.

Fig. 3. The single pedicled flap as proposed in my original contribution. Note the lateral defect left by suturing the flap to the opposite edge of the wound.

of transferring the fat lined flap to the midline, was thus located over the soft part of the buttocks with the base occupied by the gluteus maximus muscle. This defect was left to granulate and to fill in with scar tissue. The scar was thus transferred from over the long sacrum to lie over the soft gluteus maximus muscle.

While this procedure proved useful, we have modified the operation so that the technique is simpler and the results are more satisfactory (Fig. 4).

Instead of producing a pedunculated flap to be transferred to the midline, as shown in Figure 3, an incision is made lateral and parallel to one of the margins of the cavity, after the sinus has been removed. This incision is carried down to the fibers of the gluteus maximus muscle. The incision is made far enough out from the wound edge so that a wide bridge of skin and fat is freed (Fig. 4, a). The lateral incision is made long enough so that the bridge of skin and fat can be readily approximated to the opposite edge of the wound. The fat underneath and lining the bridge of skin is completely detached from the underlying gluteus maximus (Fig. 2, c), so that the entire fat lined bridge of skin can be shifted over to the midline and held there by lateral approxi-

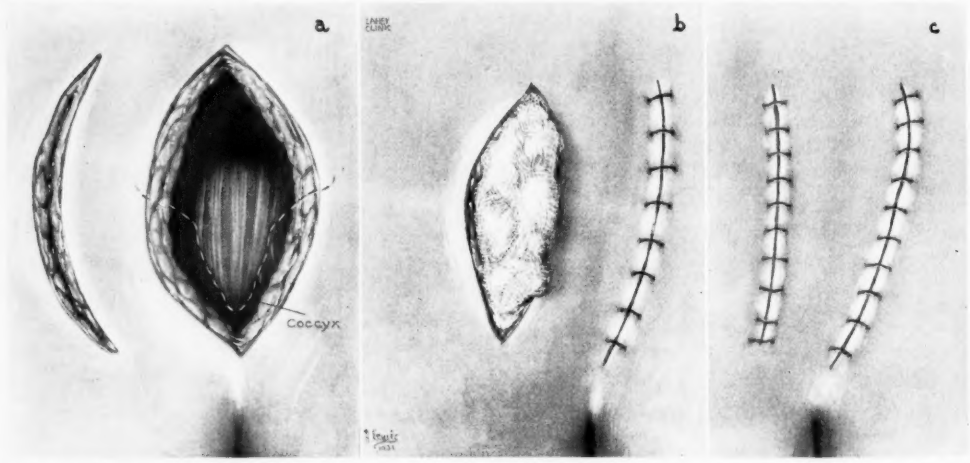

Fig. 4. Diagrammatic drawing showing large defect left after the bloc removal of the sinus and its tracts. The position of the coccyx and sacrum is shown in dotted lines. The lateral incision has been made parallel to one edge of the wound. Beneath this a bridge of skin and subcutaneous fat has been separated from the gluteus muscle so that the flap moves freely to the opposite side. In b, the fat lined bridge of skin has been displaced to the midline and sutured to the opposite wound edge to hold it in place. In this illustration the defect resulting from transference of the flap has been packed with gauze and is to be allowed to close by granulation. In c, the free bridge of skin with its fat lining has been transferred to the midline and sutured to the opposite edge of the wound. The resulting defect was sufficiently small (as occasionally happens) that its edges could be brought together and an attempt made to get healing by first intention. This procedure should not be applied in the presence of infection and, even in clean wounds, infection will occur and secondary suture of the central wound edge to its opposite edge be necessary to keep the fat lined flap in its midposition over the long sacrum.

mation stitches, the fat lined bridge of skin thus resting directly over the sacrum and providing a soft, well nourished pad of tissue to withstand the pressure of sitting.

As in the first procedure, the lateral defect left by moving this bridge of skin and fat to the midline may be closed by subcutaneous and skin approximation stitches, if it seems feasible, or the defect may be left open to granulate and fill in with scar tissue. When the scar filled defect is located laterally and over the large, soft gluteus maximus muscle, as it is in this plan, pain and pressure necrosis will not result.

The transference of a bridge of skin and subcutaneous fat nourished through attachment at both ends is superior to the pedunculated flap with a single area of attachment as used in the older method, because the doubly attached flap is better nourished and therefore is better able to combat infection which so frequently complicates an operation on a pilonidal sinus, itself so often the site of infection. We have also found that necrosis of the flap does not occur when this method is used and that secondary sutures to hold the flap in the midline may be applied, if necessary, even in the presence of a granulating wound.

CONCLUSIONS

Wide dissections and complete removal of pilonidal tracts are necessary in order to prevent the recurrence of pilonidal sinuses.

The doubly attached flap here suggested has proved superior to the flap attached at only one end as previously suggested.

FIBROBLASTOMA (FIBROMA) OF THE KIDNEY

A Report of A Case With a Review of the Literature[1]

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SMALL fibromata under the capsule, in the cortex, or in the medulla at the bases of the pyramids are not infrequently found in the routine examination of the kidney at autopsy, but they seldom cause signs or symptoms during life. According to Ewing, these fibromata have their origin in disturbances of development. Genewein, quoted by Ewing, is of the opinion that they are not true neoplasms but tumor-like nodules arising from superfluous tissue (Albrecht's hæmatoma).

In a very few cases recorded, however, renal fibromata have attained a size large enough to be of clinical significance. Because of the great rarity of this type of renal tumor, it seems desirable that the following case be placed on record.

CASE REPORT

I. W., male, aged 38 years, was admitted to the Presbyterian Hospital, July 28, 1930. Six years previous to the admission to the hospital, the patient passed gross blood in the urine. One week later he had an attack of severe pain over the region of both kidneys. Several such attacks occurred, and about 2 months later a stone was passed. Eight days before admission to the Presbyterian Hospital, the patient noticed blood in the urine and consulted his family physician who confirmed the presence of blood in the urine. There were no urinary symptoms other than the presence of painless and symptomless hæmaturia to account for the urinary disturbance.

Physical examination revealed the following: The pupils were equal and reacted to light and accommodation. The teeth showed much dental repair. The tonsils were negative. The heart and lungs were negative. The liver was palpable at the costal margin; the spleen and kidneys not palpable. The external genitalia were negative. Rectal examination was negative. Blood examination showed red cells, 5,000,000; leucocytes, 11,450; hæmoglobin, 85 per cent. The blood pressure was systolic, 131; diastolic, 80. Blood chemical examination showed urea nitrogen, 12.9; uric acid, 3.8; creatinin, 1.4; non-protein nitrogen, 35.5. Blood Wassermann test was negative. Roentgen-ray examination was negative. Urinalysis disclosed albumin, 0; sugar, 0; blood, 0; casts, 0; leucocytes, 0. Cystoscopic examination showed a normal bladder. Both ureters were catheterized without difficulty or obstruction. Examination of the urine obtained at this time showed 18 leucocytes per cubic millimeter from the bladder, 62 from the right, and 75 from the left kidney. The specimens from the bladder and both kidneys were sterile and negative for tubercle bacillus. In the phthalein test in the first half hour, the output was 60 cubic centimeters or 54 per cent; in the second half hour, 175 cubic centimeters or 14 per cent; and in the third half hour, 80 cubic centimeters or 24 per cent. Pyelograms were made: the left was normal. The right pyelogram (Fig. 1) showed a filling defect compatible with tumor. Diagnosis: tumor of the right kidney. Nephrectomy was advised.

Operation (ethylene anæsthesia) was done July 30, 1930, and consisted in a right nephrectomy. Patient made an uneventful recovery and left the hospital on August 13, 1930.

Gross and microscopic description of specimen (Dr. E. R. LeCount). The kidney was about normal size, 12 by 6 by 5.5 centimeters. The pelvis was distended by a tumor 43 by 41 by 35 millimeters, and shaped a little like a thick lens. The attachment of the tumor occupied about one-sixth of its outside and was almost altogether to the ventral half of the kidney. It was a loose attachment, close to the large renal vessels at the root of the kidney, and all around the tumor there was peripelvic fat (Fig. 2).

A thin capsule, less than a millimeter thick, surrounded the tumor and in sections made by cutting the structure was a radial one; fine wavy lines extended from all parts of the periphery toward the center. The growth was a compact, firm, and gray-white tissue. There was very little compression of the renal substance by the tumor. Most of the lining of the pelvis was red with hæmorrhage.

Histology: In sections from the deeper portions, sections equal to about 20 square millimeters, two large and several small pale spots with poorly defined outlines were present; the rest of such sections were more solidly pink (hæmatoxylin and eosin). These pale places (Fig. 3) were regions where œdema had so split apart the various constituents of the growth that the condition was virtually one of exquisitely teased-out tissue in which large fibroblasts and collagen fibrils, with occasional lymphocytes, prevailed.

The denser places with no œdema presented the usual structure of fibroblastomata, with a great variety in the quantity of collagen. Where this material was most abundant, the bands, somewhat narrower than skeletal muscle cells, were 0.35 millimeter long. Many of the blood vessels in the growth had a wall of a single layer of cells (Fig. 2) differing in no marked way from the fibroblasts. The nuclei of the fibroblasts, seen from the flat sides (Fig. 4) were 8 to 16 microns in diameter, the average about 11 microns. The chromatin was in fine granules, like dust, the nucleoli were small and frequently two in number; the nuclear membranes were very thin.

The cytoplasm for these nuclei was rarely visible, but when found was irregular and spread out as broad masses about the nuclei, polygonal and with pointed corners. Where the cells were in profile (Fig. 4), the cytoplasm was more readily seen. In such cells it extended only a short way beyond the nuclei and tapered out to two sharp points opposite one another at the poles of the nuclei. For some such cells in profile, the cytoplasm was found at only one end of the nuclei. Except where the œdema was very marked the cells were in intimate contact with collagen fibrils.

In places at the periphery of the growth, the collagen was very abundant; the fibroblasts were few and far apart. There were only a few very delicate strands of elastic tissue present, and apparently there were no smooth muscle cells whatsoever in the tumor.

[1]Presented before the meeting of the American Association of Genito-Urinary Surgeons, Buck Hill Falls, Pennsylvania, June 4-6, 1931

Fig. 1. Right pyelogram showing a filling defect compatible with tumor.

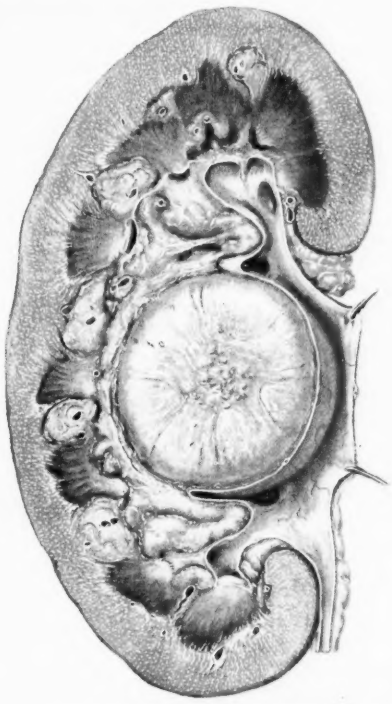

Fig. 2. Showing the presence of the fibroma with compression of the pelvis.

Large renal fibromata are also extremely rare, and on account of their rarity they may be considered curiosities both from a clinical and pathological standpoint.

In his *Monograph on Surgery of the Kidney and Ureter*, Israel states that seldom indeed do fibromata require surgical treatment. Albarran and Imbert, in a study of 529 cases collected from the literature, found two renal fibromata. Kukudshanoff, in a series of 78 cases of renal tumors reported from the Clinic of Feforow over a period of 25 years, found 1 fibroma. Hyman found no fibromata in a group of 99 tumors. Nichols presented a series of 47 renal tumors, 5 of which were benign, but there were no fibromata in this group. MacKenzie and Parkins, in a series of 56 renal tumors in adults, found 13 to be benign, including cysts, and 2 fibromata. Eusterman found no instance of such tumors in a series of 56 neoplasms. Baumgartner, in a series of 400 autopsies, found 5 cases of small nodules of fibrous tissue in the kidney which had caused no symptoms during life, and Brites found 22 such instances in 119 postmortem examinations.

HISTORICAL REVIEW

The recorded cases of pure fibromata of the kidney are, indeed, few and the literature regarding them is scanty. Dickinson, in 1862, reported to the London Pathological Society, the finding of a "fibro-fatty" tumor of the kidney. Morris states, however, that in later years, with improved pathological technique, Dickinson re-examined the neoplasm and decided that it was a sarcoma. Brodeur, in 1886, in his book on the *Surgical Conditions of the Kidney*, includes a chart of 10 benign renal tumors that he gathered from the literature, and of these 5 cases (Bruntzel, Thomas, Wahl, Claus, Park) appear to be pure fibromata. In 1896, Kuster stated that at that time there were 8 reported cases of renal fibromata. He does not, however, give the names of the authors, dates, or places of reports of these tumors.

In addition to the cases reported by Brodeur, cases of renal fibromata have been reported by Wilks, Clark, Steinle, Boross, and Kukudshanoff. Thus, with the addition of the case reported in this paper, there are available for study records of 11 cases of pure renal fibromata.

Great care has been taken to exclude from this series the numerous cases of tumors which con-

FIBROMA OF THE KIDNEY

No.	Author	Age	Sex	Chief symptoms	Duration	Clinical diagnosis	Treatment	Location	Outcome	Comments
1	Wilks 1869	53	M	Intermittent pain in loins and hæmaturia Abdominal tumor right side	10 yrs. 6 yrs.	Kidney stone or cancer	No operation Autopsy specimen	Right kidney	Death with uræmic symptoms	Tumor discovered at autopsy. Covered by kidney capsule which it had gradually distended. Destruction entire kidney substance by compression.
2	Bruntzel 1882	33	F	Large abdominal tumor	5 yrs.		Nephrectomy	Left kidney	Recovery	Tumor arose from kidney capsule—weighed 20 pounds.
3	Thomas 1882	21	F	Abdominal tumor size of 8 mos. pregnant uterus		Ovarian tumor	Nephrectomy	Lower pole left kidney	Recovery	Tumor arose from kidney capsule. It showed much cystic degeneration.
4	Claus 1885	46	F	Abdominal tumor right side		Ovarian cyst	Nephrectomy	Right kidney	Recovery	Tumor developed between cortical and medullary portions.
5	Wahl 1885	11		Abdominal tumor	6 yrs.	Benign renal tumor	Nephrectomy	Lower pole of right kidney	Recovery	Tumor arose from kidney capsule. Shortly before tumor was noticed received trauma to abdomen (fall).
6	Park 1886	2		Enlargement right side of abdomen, palpable tumor	Since few mos. after birth	Tumor of right kidney	Nephrectomy	Right kidney	Good condition 7 mos. later	Weighed 4 pounds. Showed extensive cystic changes.
7	Clark 1923	28	F	Pain (right side). Mass size of tennis ball (right kidney region).	1 yr.	New-growth of some kind	Nephrectomy	Right kidney	Condition good 13 mos. later	No urinary disturbances were shown.
8	Kukudshanoff 1928	36	F	Anæmia and large palpable left kidney	5 yrs.	Neoplasm of left kidney	Nephrectomy	Left kidney	Recovery	Tumor imbedded in kidney and revealed by sectioning. It measured 12 by 8 by 6 cm.
9	Boross 1929	23	M	Hæmaturia			Nephrectomy		Recovery	Pediculated in kidney pelvis.
10	Steinle 1929	28	M	Left lumbar pain Profuse hæmorrhage from left ureter, renal colic	6 wks. Shortly before operation	Renal neoplasm	Nephrectomy	Left kidney, upper pole	Recovery	
11	Kretschmer 1931	38	M	Hæmaturia was the only complaint	8 das.	Tumor of right kidney	Nephrectomy	Right kidney in hilus outside but adjacent to pelvis	Recovery	Gave history of having passed a stone about 6 yrs. previous to coming under observation.

Fig. 3. In the paler regions the fibroblasts and cells are spread apart by œdema. The darker half is mainly collagen fibers stained with phosphotungstic acid hæmatoxylin.

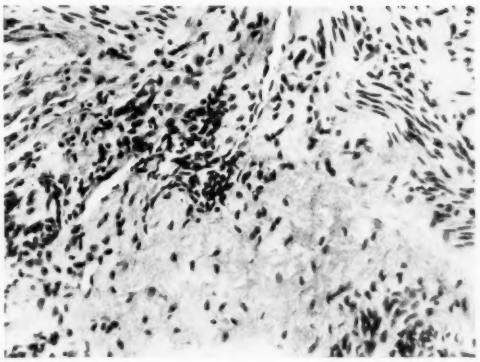

Fig. 4. A collapsed empty blood vessel is in the center of the top. On the left side of this are many fibroblasts with the nuclei flat, seen from the sides, so they appear oval and faintly stained. On the right of the vessel the nuclei of many fibroblasts lie edgewise, are darker, slightly wavy, and in places parallel.

tain other elements than fibrous connective tissue (chondro-fibromata, osteofibromata, fibrolipomata, fibro-adenomata, etc.). Nor was it possible to include those fibromata which are merely mentioned in the classification of types in series of renal neoplasms, to the effect that in a given series a fibroma was found but the clinical data were lacking.

PATHOLOGY

Renal fibromata may take their origin in any part of the kidney. In 3 cases the tumor had its origin in the kidney capsule; in 1 case in the upper pole; in 1 case the growth gradually distended the kidney capsule, destroying by pressure the kidney substance but preserving in general the normal kidney contour; in 1 case the tumor developed between the cortical and medullary portions; in 1 case the tumor, which was pediculated, hung into the renal pelvis; in 1 case the tumor was "embedded" in the kidney; in my case the tumor occupied the middle third of the kidney, in the ventral half; in 2 cases, the origin was not given.

These fibromata have been described as having the same firm consistency and homogeneous, grayish-white appearance typical of this type of tumor. They are frequently mentioned as having undergone mucoid and pseudocystic degeneration in portions. Microscopically, they are found to consist of fibrous connective tissue but they show no special characteristics.

The right kidney was the site of the growth in 5 cases and the left in 3 cases; in the case herein reported the tumor was located in the right kidney and this information was not given in the other reports.

ETIOLOGY

With regard to fibromata of the kidney, Ewing states that "these growths must owe their origin to some developmental disturbances of the kidney." In this regard we may mention that in the cases reported by Park and Wahl, the tumors occurred in children of 2 and 11 years respectively.

Garceau is of the opinion that the fibromata which become important clinically have their origin in the small fibrous nodules which have previously been mentioned and are not infrequently found at autopsy. These nodules "may become active and attain a large size."

AGE

The youngest age of occurrence in this series was 2 years (Park) and the oldest 53 years (Wilks). In 2 cases the growth occurred before the age of 20 years, 4 between 20 and 30, 2 between 30 and 40, 1 between 40 and 50, and 1 after 50 years. The patient here reported was 38 years of age.

SEX

The case reports did not always mention the sex. In the series there were noted 5 females and 4 males, including my case.

SYMPTOMS

As observed by Stillman, Morris, Clark, and others, renal fibromata, until they have attained

a large size, usually cause neither signs nor symptoms by which they render themselves apparent clinically. In 8 of 11 of the recorded cases the presence of an abdominal tumor served to direct the patient's attention to the condition. The tumors vary in size from that of a tennis ball (Clark) to that of an 8 months' pregnancy (Thomas), and in some instances the abdomen from the xiphoid to the brim of the pelvis is filled (Bruntzel).

Pain is a frequently mentioned symptom and was present in the cases of Clark, Steinle, and Wilks. MacKenzie and Parkins state that pain and the palpable abdominal tumor were present in both their cases.

Many of the authors mentioned the fact that the urine showed no abnormal findings and the patient was free of urinary disturbances. However, in my case the patient had an attack of profuse painless hæmaturia and it was for this reason that he consulted a physician.

Steinle's case is unusual in that it too presented a profuse renal hæmorrhage and severe renal colic. Intermittent hæmaturia over a period of 10 years was present in Wilks' patient. MacKenzie and Parkins remarked that hæmaturia was never present in their 2 cases and they suggested that the lack of hæmaturia might serve as a distinguishing feature to differentiate this benign tumor from a malignant growth.

In 2 cases, renal fibromata were incorrectly diagnosed as ovarian cysts; in 7 cases the diagnosis was renal neoplasm; and in 2 cases, the neoplasm of the kidney was not diagnosed.

One outstanding feature in the history of these tumors is their long duration; 5 years in Bruntzel's case, 6 years in Wahl's, and 10 years in Wilks'. In Steinle's case, which came to operation within 6 weeks after the onset of symptoms, the operation being preceded by severe hæmorrhage and renal colic, the acute symptoms were more pronounced than in any of the other recorded cases.

In the earlier cases reported, the patients of course did not receive the benefit of the modern means of urological diagnosis; for example, cystoscopy, ureteral catheterization, and the pyelogram. In my patient, who was more fortunate in this respect, the pyelogram showed a filling defect compatible with tumor, but gave no clue regarding the nature of the tumor.

TREATMENT

Nephrectomy was done in 10 of the 11 cases in this series. If it were possible to determine that, in a given case, the tumor is benign, the growth alone might be removed and the patient spared a nephrectomy.

PROGNOSIS

From a study of the literature it would seem that the prognosis in fibromata of the kidney is extremely favorable. A favorable outcome has been reported in every instance except in Wilks' case in which, in spite of the fact that for more than 6 years it was known that an abdominal tumor was present, surgical intervention was not attempted and the patient died of symptoms of uræmia. MacKenzie and Parkins state that the outcome in their cases of renal fibromata was favorable. Brodeur and Morris believe that this type of tumor is benign, clinically and pathologically.

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THE DEVELOPMENT OF THE MERCUROCHROME TECHNIQUE IN OBSTETRICS

A Report of Ten Thousand Cases, Five Thousand of Which Were Studied During the Experimental Stage

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ONE of the greatest problems that confronts the medical profession of this country today is to find means to reduce maternal morbidity and mortality. A large sum of money has been given in order that a study may be made of puerperal deaths in New York City and with the hope that the reason for the mortality rate may be determined. The medical as well as the lay press publish hundreds of articles deploring the present situation; physicians are criticized and statements have been made that the expectant mother is safer in the hands of a midwife than surrounded by the latest hospital methods of combating shock, hæmorrhage, and the accidents incident to childbirth.

I believe that it is generally admitted that we have made progress in the treatment of toxæmia, contracted pelvis, and postpartum hæmorrhage and that therefore a patient with any of these complications is far safer in the hands of a physician. It is also true, however, that when it comes to the prevention of deaths from puerperal septicæmia, year after year brings the same grand total. Would it not be well for us to stop to consider what we are doing to combat this ever present condition? A brief survey of the routine perineal and vaginal preparations as practiced in the majority of hospitals will soon convince us that the methods used to prevent "child-bed fever" are little better than they were 25 or even 50 years ago. To my mind, there is little use in being careful to sterilize drapes, instruments, gloves, etc., only to leave undisturbed the vagina with its sacred pathogenic organisms.

The medical profession as a body is hard to convince. What they were taught and what was good enough for them during the last 25 or 50 years is still good enough. One doctor will say that he has been doing obstetrics for 10, another for 15, and still another for even 25 years, and that he has never lost a case from puerperal sepsis. This may be true, but it is no reason why he should not advocate or teach the sterilization of the vagina especially when apparently all other methods and teachings have failed to reduce the death rate from puerperal sepsis.

Some claim that the great drawback to vaginal antisepsis is that it may lead to more meddlesome midwifery than at present. We have about reached the highest pinnacle in meddlesome midwifery. If the doctor would examine his patient more carefully and not rely on rectal and abdominal examinations, he would be not only a better judge as to whether a patient is abnormal, but undoubtedly he would be a better obstetrician. Unquestionably cæsarean sections would be less frequently done if patients were more carefully examined, if doctors were not so fearful lest their patients become potentially infected, and if the patient were given a test of labor before being subjected to the ordeal of cæsarean section.

In an article entitled "Mercurochrome as an Antiseptic in Obstetrics," Baldwin reports a series of 61 cases in which he used 1 ounce of a 2 per cent solution of mercurochrome in the vagina every 6 hours during labor, with a morbidity of 13.4 per cent, as compared with 80 cases in which no mercurochrome was used, with a morbidity of 22.5 per cent. He states that "mercurochrome has not reduced maternal morbidity to any marked degree." I am convinced, however, that if his series were larger and the technique properly carried out, his results would be more strikingly favorable.

Henderson showed a reduction of 42 per cent in the maternal morbidity of 100 consecutive cases in which mercurochrome was used, when compared with 100 cases without mercurochrome. He states: "There is a certain added sense of security which this procedure gives to the mind of the obstetrician, especially in those more critical and difficult situations."

Brown reports a series of 5,385 deliveries in which he used a solution containing 15 grams of mercurochrome crystals, 5 cubic centimeters of one-half strength iodine, and 500 cubic centimeters of glycerine, with 11 deaths from puerperal infection, 7 of which were infected before admission to the hospital. He compares this with 2,194 cases with 10 deaths, 5 of which were infected before admission to the hospital, the percentage being 0.45 without sterilization of the

vagina and 0.20 with sterilization. He states that "anaerobic streptococcal puerperal infections will perhaps be best reduced by using some antiseptic preparation in the vagina at the beginning of, and during, labor. At the present time we recommend no particular preparation, but hope to determine by experience and experimental work what solution will prove most efficacious. We predict that this subject will be one that will command one of the most important investigations of the existing problems of modern obstetrics."

In an article entitled "Puerperal Morbidity without Disinfection of the Vagina," Gordon reports 2,016 cases with a gross morbidity of 3.6 per cent. This is certainly an enviable record, especially when we consider the fact that the majority of the patients were delivered by the interne staff. He recommends the application of a 3.5 per cent tincture of iodine solution in the preparation of the perineum. According to the work of Raiziss and his collaborators, a 2 per cent aqueous solution of mercurochrome is preferable to a 5 per cent tincture of iodine, as positive growths were obtained in all tests when the iodine was allowed to come in contact with the skin for 15 minutes, while he reports 33 per cent negative cultures with mercurochrome and with the acetone mercurochrome, only a 2 per cent solution being used, 87 per cent negative. Also if the 2 per cent solution of mercurochrome was allowed to come in contact with the skin for an hour, all the tests were negative, while with the iodine, no negative tests were obtained. Patients object strenuously to the irritation and burning from the iodine; it is an irritant to the skin of the perineum and the sensitive mucous membrane of the vagina. Gordon admits that infection may occur from within or without, but doubts that the reduction of the number of bacteria in the vagina would minimize the risk.

The possible value of vaginal antisepsis was first brought to my attention during the month of February, 1924. Mrs. M. had had a normal delivery and was returned to her bed. Following the delivery of her first child, 2 years previously, she had had a postpartum hæmorrhage which was checked with difficulty. Realizing this and in view of the fact that she continued to bleed, we deemed it necessary to pack the vagina. I decided to use a 2 per cent aqueous solution of mercurochrome for the perineal preparation. This was applied only to the perineum and the vagina was packed with plain gauze. This patient should have had a normal convalescence, but she developed a postpartum infection, was in the hospital for 75 days and after returning home she was an invalid for months. The question arose as to why this patient developed a postpartum sepsis. Sterile gloves, drapes, instruments, etc., had been used and the perineum had been carefully disinfected, but we had left the vagina absolutely alone. This omission undoubtedly was the cause of her protracted illness. The outcome in this case was the incentive for beginning the use of mercurochrome as a vaginal antiseptic in the Methodist Episcopal Hospital.

DEVELOPMENT OF THE MERCUROCHROME TECHNIQUE YEAR BY YEAR

1923

Technique: The perineum and surrounding area were painted with 3.5 per cent iodine at the time of delivery. Lysol was used to flush the perineum.

Morbidity: There were 877 vaginal deliveries with a morbidity of 12.5 per cent (Chart 1).

1924

Technique: Same as for 1923 with the exception that 268 cases had the perineum sprayed with mercurochrome and mercurochrome was used in the vagina at the time of delivery.

Morbidity: 1,195 deliveries with a morbidity of 12.3 per cent.

1925

During 1924 the outcome of the mercurochrome technique did not seem so brilliant and it was thought that our results might be better if we instilled the vagina at the beginning of labor.

Technique: Beginning February 20, 1925, a small rubber tube, 4 inches in length, attached to a one-half ounce asepto syringe, was used to instill the patient at the time of admission to the hospital and every 8 hours during labor.

Morbidity: During this year 1,168 patients, exclusive of those who had cæsarean sections, were delivered following the mercurochrome technique, with a morbidity of 6.8 per cent. These results showed a 44 per cent drop in the morbidity when compared with the year 1924.

Publication: During 1925 the first article was published on "The Use of Mercurochrome in Obstetrics." It included a report on 368 cases, in which the mercurochrome technique had been used, with a morbidity of 12.2 per cent. This was compared with 410 cases, in which the iodine technique had been used, with a morbidity of 10.8 per cent.

1926

We were not satisfied with our results during 1925 and thought perhaps it might be better to instill all of the patients at the time of vaginal examinations. This would do away with the rectal examinations during labor and was an experiment on our part to test out this method of instillation.

Technique: Two fingers were inserted well into the vagina, the pelvic floor was depressed, and a dram of mercurochrome instilled and worked into the folds of the mucous membrane and around the cervix or presenting

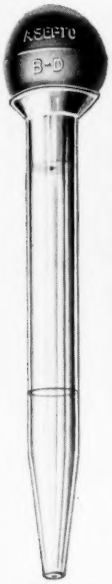

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 1. Syringe which we have used for the vaginal instillation of mercurochrome for the last 5,000 cases. We attribute our success with the mercurochrome technique to the use of this syringe.

Fig. 2. Roentgenogram taken following instillation of the vagina with a catheter and small syringe. An opaque solution was used instead of the mercurochrome. The vagina is not distended and the instillation by this method is unsatisfactory.

Fig. 3. The result when the proper instillation of the vagina is done by means of the asepto vaginal syringe. The vagina is ballooned so that the rugæ are visible and the mercurochrome is forced into the cervix and folds of the vagina.

part. The instillations were done at least every 8 hours, but frequently, in order to follow the course of labor, they were repeated oftener.

Examinations: From 1923 to 1926 the progress of labor was followed by the use of rectal examinations, and vaginal examinations were done only when indicated. Beginning January 1, 1926, all cases were followed during this year by the use of vaginal examinations alone.

Morbidity: During this year there were 1,740 deliveries with a morbidity of 9.2 per cent.

Publication: An article entitled "Morbidity in Obstetrics—Its Reduction by the Use of Mercurochrome as a Vaginal Antiseptic" was published during this year and reported 1,118 cases following the mercurochrome technique, with a morbidity of 6.8 per cent, as compared with 2,072 cases without mercurochrome, with a morbidity of 12.4 per cent.

1927

The very apparent increase in morbidity during 1926 over the previous year was possibly explained by the fact that although it is possible to do the instillation properly at the time of vaginal examinations, this method as a routine is not entirely satisfactory because the examining fingers of one doctor may be properly adapted for doing pelvic examinations while others, because of their size or shape, may be improperly suited.

Technique: We were convinced that this fact increased our morbidity during 1926 and at the beginning of 1927 we went back to the catheter and small syringe method of instillation as carried out during 1925.

Examinations: Rectal examinations were routine and vaginal examinations done only when indicated.

Morbidity: In 1,853 deliveries in 1927, there was a morbidity of 9.6 per cent.

During the spring of 1927 there were so many deaths from puerperal sepsis in New York City that we became rather discouraged because of our high morbidity and increased mortality from sepsis. However, other hospitals were reporting similar epidemics and some even being obliged to close their doors to new admissions.

Publications: An article was published during 1927 entitled "The Use of Mercurochrome in the Preparation for Delivery." This paper described a study of the different methods of preparing the external genitalia before the application of mercurochrome, with the following results. In 74 cases in which the perineum was clipped without cleansing and mercurochrome was applied, the morbidity was 6.7 per cent. In 50 cases in which the clipping was followed by cleansing with soap and water before the application of mercurochrome, the morbidity was 4 per cent. While in 262 cases in which the perineum was thoroughly prepared by shaving and cleansing, the morbidity was 9.2 per cent.

Also during this year an article entitled "The Use of Mercurochrome as a Vaginal Antiseptic in the Induction of Labor" was published, which detailed 93 cases in which

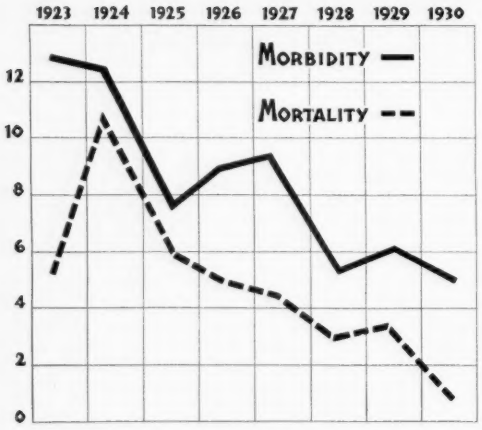

Chart 1. Comparative study of the morbidity and mortality from 1923 to August 1, 1930. The morbidity is in percentage and the mortality represents the number of deaths per 1,000 deliveries, not including cæsarean sections.

mercurochrome was not used and the morbidity was 29 per cent as compared with a morbidity of 11.5 per cent in 78 cases treated by the mercurochrome technique.

1928

It was during 1927 that we discovered that the mercurochrome did not always reach the vaginal vault when the catheter and syringe method was used. While taking a cervical culture, following the routine instillation, I found that, on introducing the speculum, the upper third of the vagina was entirely free from mercurochrome.

Technique: October 1, 1927, we changed our method of instillation, using the special asepto vaginal syringe, shown in Figure 1, for all of our instillations during the progress of labor. Also beginning October 1, 1927, the perineum was sprayed at the time of delivery with a 4 per cent aqueous, alcohol, acetone solution of mercurochrome instead of the 4 per cent, aqueous solution which we still continued to use during the progress of labor.

Examinations: The usual routine rectal examinations were made but vaginal examinations were made only when indicated.

Morbidity: 1,970 patients were delivered in 1928 with a morbidity of 5.1 per cent. This was a decided drop over any of our previous records, and we felt confident that it was due to the more thorough instillation by means of the syringe and the fact that the aqueous, alcohol, acetone solution was used on the perineum.

A large number of the patients during 1928 were examined frequently in order to secure cultures for a study of the effect of mercurochrome on the vaginal flora during labor and following delivery. Under ordinary circumstances, if the mercurochrome technique had not been used, this would increase the morbidity.

Publication: A study of the morbidity following cæsarean section was compiled during this year, 218 cæsarean sections being reported in which the mercurochrome technique was used, with a morbidity of 42.2 per cent, as compared with 120 cases in which the mercurochrome was not used, with a morbidity of 57.5 per cent.

1929

Technique: Same as that used in 1928.

Examinations: On the second obstetrical service a large number of the patients were restricted to vaginal examinations while on the first obstetrical service, rectal examinations were routine.

Morbidity: There were 1,975 deliveries with a morbidity of 6.2 per cent.

1930 (7 months)

Technique: Same as in 1928 and 1929.

Examinations: Same as 1929.

Morbidity: There were 1,155 deliveries during the first 7 months of 1930, with a morbidity of 59 or 5.1 per cent. There was only one maternal death which could be laid to sepsis, and this patient was only 5½ months pregnant, and had had chills and fever before admission to the hospital. Mercurochrome was used only at the time of delivery.

Publication: An article entitled "A Bacteriological Study of the Value of Mercurochrome as a Vaginal Antiseptic, with Particular Reference to its Use in Obstetrical Cases" showed that the number of positive cultures from the vagina was reduced from 44 per cent to 6 per cent; from the cervix, 16 per cent to 4 per cent; from the membranes, 32 per cent to 6.4 per cent.

Table I shows a detailed analysis of the deliveries, with the resulting morbidity, for the three periods (Chart 2). During the first, 1923 through 1924, before the use of mercurochrome, there were 2,072 deliveries, exclusive of cæsarean section, with a morbidity of 12.4 per cent. The years 1925 through 1927 covered the experimental period during which the best technique for vaginal instillations was being established. During this period there were 5,076 deliveries with a morbidity of 8.9 per cent. One variation in technique was made in that on the second obstetrical service a large number of the patients were subjected to vaginal examinations to determine whether or not this would influence morbidity.

It is evident, from a study of the comparative percentages for the different periods, that there has been a steady decline in morbidity in practically all conditions represented in the table. Primiparæ show a higher morbidity than multiparæ; operative deliveries, a higher morbidity than spontaneous deliveries; breech deliveries in past 3 years showed 5.9 per cent morbidity; and as for toxæmia, in 244 cases, including all degrees of toxæmia, the morbidity was 4.09 per cent.

Previous reports and publications have included all the mercurochrome deliveries when giving the percentage of morbidity. This is unfair to the technique which has been established at the present time. Many of those who have used mercurochrome in obstetrics have obtained poor results because they still follow the original technique.

With our present technique, which has been used almost 3 years, we trust that our low morbidity will continue.

The question has been raised repeatedly as to whether the instillation should be repeated every 6, 8, or 12 hours. A study of Table II will show that in 30 cases either no mercurochrome was used or instillations were made at the time of delivery. In 46 cases the mercurochrome did not remain in the vagina over an hour before delivery. In 68 patients instillations were made from 3 to 6 hours before delivery; in 33, from 6 to 9 hours; and in 17, from 9 to 12 hours. In 89 cases mercurochrome was instilled over 12 hours before delivery, and of these in 68 more than one instillation was made.

If we omit the 30 cases with a morbidity in which the mercurochrome was either not used at all before delivery or used only at the time of delivery, we would have a morbidity of 5 per cent.

The morbidity percentage does not tell the whole story. In fact, I feel that a better representation of the morbidity is conveyed when the total number of days' morbidity is enumerated, and then the average number of days' morbidity per patient, as is shown in Table III. When we first began the use of mercurochrome, it was the decrease in the days' morbidity in the mercurochrome group that was encouraging in spite of the fact that the morbidity percentage was higher with mercurochrome. If our only aim had been to send our patients home from the hospital alive, then we might say that the only morbidity worth considering was the morbidity that was severe enough to cause maternal deaths. This could very easily be corrected to the deaths from sepsis.

A study of Tables III, IV, and V is very instructive. The average days' morbidity per patient before the use of mercurochrome was 0.40, 0.46 during the experimental stage, and with the present directions carefully followed, the average days' morbidity for 5,102 cases is 0.26 days per patient. Table V shows that during 1923 and 1924, 78 patients had a morbidity of 2 days each, while in the experimental mercurochrome group, 140 patients had a morbidity of 2 days, and in the latest mercurochrome group there were 100 patients with 2 days' morbidity. In other words, if we were to omit the patients with 2 days' morbidity in the last 5,102 cases, we would have a 33 per cent reduction in the morbidity. Another very striking comparison is in the number of patients who had a morbidity of over 20 days. Without mercurochrome the ratio was 1 in 345; in the experimental mercurochrome series it was 1 in 390; while in the last mercurochrome group there were only 7 cases in 5,102 deliveries, or 1 in 728. An analysis of these is shown in Table VI.

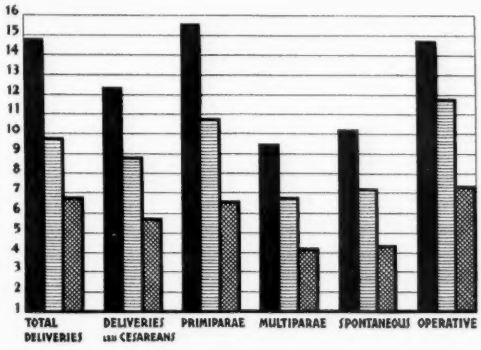

Chart 2. Comparative study of the percentage morbidity for the three groups of cases. The first bar represents the years from 1923 through 1924; the second bar, 1925 through 1927; the third bar, 1928 through July, 1930.

In 3 cases in Table VI deliveries were spontaneous. In 1 case instillation was done at the time of delivery and in another at the time of induction. Labor was short in all but 1, who developed a bilateral phlebitis. The 2 patients who developed pelvic abscesses had no vaginal examinations, very short labors, membranes intact until a short time before delivery, and both were multiparæ.

Table VII shows the total number of deliveries for each month and the percentage of morbidity. March, as would be expected, has the highest morbidity rate, but this is accounted for to a great extent by the fact that during the year 1927 we had a severe influenza epidemic with a morbidity in 28 cases. If we omit the cases delivered and the morbidity for March, 1927, it gives us a morbidity for that month, for the 4 years, of 8.1 per cent.

In the months of September and November the morbidity was comparable with that of January and February. The lowest rate was for December, a winter month, when a higher morbidity would be expected. July and August, the summer months, carried the largest number of deliveries.

A careful scrutiny of this table would lead one to believe that the season of the year has very little to do with the percentage of morbidity when thorough vaginal antisepsis is carried out.

Table VIII shows the corrected and uncorrected morbidity for the years 1928 to 1930, in which a large percentage of the morbidity is classified as being due to lochiometra and sapræmia. In these patients the morbidity was of very short duration, 2 or 3 days. The findings were generally represented by a moderate degree of subinvolution of the

TABLE I.—MORBIDITY REPORT FOR THE YEARS 1923–30

	Without mercurochrome 1923–24			Experimental stage 1925–27			Present technique 1928–30		
	Cases	Morbidity	Per cent morbidity	Cases	Morbidity	Per cent morbidity	Cases	Morbidity	Per cent morbidity
Deliveries	2196	327	14.8	5253	525	9.9	5279	364	6.8
Cæsarean section	124	69	55.6	177	73	41.2	177	77	43.5
Deliveries less cæsarean sections	2072	258	12.4	5076	452	8.9	5102	287	5.6
Ward	847	103	12.1	2080	212	10.1	2138	139	6.03
Private	1225	155	12.6	2996	240	8.01	2964	148	4.9
Primiparæ	1041	161	15.4	2587	278	10.7	2717	188	6.5
Multiparæ	1031	97	9.4	2489	174	6.9	2385	99	4.1
Spontaneous	1105	114	10.3	3232	232	7.1	3077	136	4.4
Operative	967	144	14.8	1844	220	11.9	2025	151	7.4
Low and prophylactic forceps	675	80	10.3	1412	165	11.6	1566	111	7.08
Medium forceps	179	27	15.08	250	34	14.4	317	22	6.9
High forceps	53	18	33.9	45	5	11.1	38	5	13.1
Axis traction				27	3	11.1	33	4	12.09
Barton				10	0	0	37	5	13.5
Piper				17	2	11.7	56	4	7.1
Versions	32	12	37.5	56	7	12.5	35	2	5.7
Inductions	28	7	25.	91	9	9.8	69	11	15.9
Breech	108	10	9.2	247	29	11.7	219	13	5.9
Scanzoni	114	22	19.2	93	17	18.2	32	4	12.5
Left occiput anterior	1009	127	12.5	3036	247	8.1	3062	119	3.8
Right occiput anterior	399	35	8.7	1022	77	7.5	922	55	5.9
Left occiput posterior	121	24	19.8	339	33	9.7	317	22	6.9
Right occiput posterior	184	28	15.2	361	35	9.6	429	30	6.9
Placenta prævia	19	9	47.3	41	10	24.3	37	7	18.9
Eclampsia	9	2	22.2	25	10	40.	22	3	13.6
Toxæmia	39	3	7.6	189	26	13.7	244	10	4.09
Lateral episiotomy	66	19	28.7	1547	166	10.7	2447	171	6.9
Median episiotomy	821	107	13.03	811	67	8.2	530	23	4.3

uterus, and at times, the presence of foul lochia.

It will be noted that during this period there was no note on the chart of 39 of the patients as to the cause of the morbidity or the examination was negative. In many of these patients the temperature would reach 100.4 degrees on 2 days only and was overlooked by the man signing the chart.

In those cases in which morbidity was not due to delivery, respiratory conditions were supposed to account for the condition in 42 cases, while the breasts were blamed in 40; pyelitis accounted for 35. Twenty occurred during 1929 and only 5 during the year 1928.

THE STANDARD OF MORBIDITY

When we consider the different standards of morbidity, it is very hard to say which one is right. It would seem better to accept the one which is most universally adopted. There are undoubtedly a great many maternity patients who have some rise in temperature during the puerperium which means little or nothing as far as their well-being is concerned. There are others who have a prolonged morbidity and they may not die, but are left invalids for months or perhaps for life and still they are classified with the other patients who have a rise in temperature to, say,

TABLE II.—HOUR OF INSTILLATION

	Morbidity Cases, 1928–30
At delivery	15
No mercurochrome	15
Mercurochrome given less than 1 hr. before delivery	16
Mercurochrome given 1 to 2 hrs. before delivery	15
Mercurochrome given 2 to 3 hrs. before delivery	28
Mercurochrome given 3 to 4 hrs. before delivery	30
Mercurochrome given 4 to 5 hrs. before delivery	17
Mercurochrome given 5 to 6 hrs. before delivery	21
Mercurochrome given 6 to 9 hrs. before delivery	33
Mercurochrome given 9 to 12 hrs. before delivery	17
Mercurochrome given 12 hrs. or over before delivery	21
Mercurochrome given 2 times	50
Mercurochrome given 3 times	18

TABLE III.—A STUDY OF THE DAYS' MORBIDITY

	1923–24	1925–27	1928–30
Total days' morbidity	2072	2352	1355
Average days' morbidity per patient	0.40	0.46	0.26

100 degrees for a single day during the puerperium. What is the use of reporting the morbidity in a group of 100 cæsarean sections and saying that all but 10 or perhaps 15 had a morbidity? I am of the opinion that the standard which we have adopted at the Methodist Episcopal Hospital is too stringent in such cases and hence they are given separate from vaginal deliveries. In a large number, this rise in temperature is a reaction which should not be considered a morbidity.

We have accepted the standard of the American College of Surgeons and also the Congress of Vienna; namely, a rise in temperature to 100.4 degrees on 2 consecutive days, not including the first 24 hours and occurring on or before the tenth day. If the patient has a rise to 100.4 degrees on the tenth and eleventh days postpartum, we consider it a morbidity. I understand that in some clinics if the temperature does not rise to 100.4 degrees until the tenth day, it is not included.

The interpretation which we put on this definition may change the percentage of morbidity. If we would say, for instance, that the temperature had to stay above 100.4 degrees for 24 hours, then the morbidity which I have given would be reduced over 50 per cent, because nearly all patients, even with a temperature of 103 degrees on 2 consecutive days, will invariably drop below 100.4 at some time during the 24 hours. As I understand the exact reading of the report from Vienna, this is the interpretation we should put upon it. Then again, can you imagine what effect it would have on the morbidity if the temperatures were taken carelessly, if the temperature were charted without being taken, or perhaps the temperature taken only in the morning and not in the afternoon, when it is likely to be the highest? I do not believe

TABLE IV.—A STUDY OF THE DAYS' MORBIDITY

	1923	1924	1925	1926	1927	1928	1929	1930
Total deliveries less cæsarean sections	877	1195	1483	1740	1853	1878	1975	1157
Total days' morbidity	335	499	433	811	1108	472	532	351
Average days' morbidity per patient	.38	.41	.23	.46	.54	.23	.27	.30

TABLE V.—DAYS OF MORBIDITY

Days	1923–24	1925–27	1928–30
2	78	140	100
3	51	92	67
4	36	64	36
5	20	36	33
6	7	26	12
7	12	20	10
8	7	14	6
9	3	13	5
10	2	3	6
11	4	5	2
12	2	3	2
13	4	2	1
14	0	2	0
15	3	4	0
16	1	0	1
17	0	2	0
18	0	0	1
19	0	1	1
20	6	13	7

that it is necessary to take the temperature every 4 hours during the entire stay of the patient in the hospital, but we believe that it is essential the first 8 or 9 days, and thereafter it should be taken at least twice a day, one of the temperatures being recorded at 4 p.m., and if at any time the temperature reaches 100 degrees, it should be taken every 4 hours.

Mouth temperatures have been the routine at the hospital, except in operative cases. When the temperatures are taken by rectum, they are, of course, higher than when taken by mouth. We have frequently checked the mouth and rectal temperatures in the presence of a morbidity and found a distinct variation, but it was seldom as much as one-half of a degree and at times they were identical. The question arises whether or not we should consider such temperatures a morbidity. We have determined our morbidity by the record as charted on the temperature sheet.

I made a study of 100 consecutive charts and found that 46 had at least one rise of temperature to 99 degrees; 16, to 100 degrees; 10, to 100.4 degrees; while if we use the standard of the American College of Surgeons, we have a morbidity of 2 per cent. If a temperature of 100.6 degrees was used instead of 100.4 degrees, the morbidity was the same, but if we required the temperature to stay over 100.4 degrees for 48 hours, the morbidity was zero in each case.

TABLE VI.—PROLONGED MORBIDITY

Hospital number	Para	Days' morbidity	Examinations		Mercurochrome	Membranes	Duration of labor	Delivery	Cause of morbidity
			Rectal	Vaginal					
6002	V	61	0–0		5 hrs.	18 min. before delivery	7 hrs. 36 min.	Spontaneous, left occiput anterior No laceration	Pelvic abscess.
7927	I	32	6–1		Times 3	35 hrs.	35 hrs.	Medium forceps, left occiput anterior	Bilateral phlebitis.
6215-A	II	21	0–1		2 hrs.	0	2 hrs. 24 min.	Spontaneous, left occiput anterior	Tuberculosis of lungs.
9181	I	22	2–8		Times 3	53 min.	13 hrs.	Prophylactic forceps, right occiput transverse	Bronchial pneumonia. Infected perineum.
9537	II	28	0–1		At induction	0	1 hr. 15 min.	Prophylactic forceps Face presentation Bag induction	Encephalitis. Chorea.
9685	VIII	46	0–0		At delivery	42 min.	57 min.	Bag induction. Placenta prævia. Breech	Pelvic abscess.
9772	I	26	2–0		3 hrs.	10 hrs.	10 hrs.	Spontaneous right occiput anterior	Abscess of leg. Mastitis.

At the beginning of this work at the Methodist Episcopal Hospital, I made very little effort to correct the morbidity because many of these cases were not recognized as morbidities by the attending physicians and so they made no note on chart. Thus it was impossible to correct the morbidity.

TECHNIQUE

Preparation on admission. The pubic hair is shaved off and the perineum and surrounding field are cleansed with green soap and water, making sure that all sebaceous material is removed from the labial folds.

The external genitalia and surrounding area are sprayed with a 4 per cent, aqueous solution of mercurochrome. We have been using a De Vilbiss atomizer for the spraying of the mercurochrome solution, but we find considerable difficulty in keeping the atomizer working properly. The spray is too fine and unless considerable care is taken, the area is not properly covered. I have been endeavoring to get a better-suited atomizer for this purpose. At all events the atomizer should be cleaned daily and if a stillette is kept in it when not in use, it will aid considerably. If the atomizer does not work properly, a stick sponge saturated with mercurochrome can be used to paint the perineum.

The asepto vaginal syringe (Fig. 1) that we are now using should have an outside diameter of 7/8 inch and a barrel 7 inches long, with a circular mark to indicate a capacity of 3 drams. The syringe should be filled with mercurochrome to this point. Then, the labia are separated and the point of the syringe is passed along the vaginal floor until it reaches the vault of the vagina. After the syringe is inserted properly, the labia should be held close together around the syringe with the thumb and finger of a gloved hand. This keeps the fluid from leaking when the bulb is pressed, causes the fluid to enter the vagina under slight pressure and insures its coming in contact with the entire vaginal mucosa. Figure 2 represents the amount that would come in contact with the vaginal mucosa when the instillation is done by means of a catheter and small syringe. This method was used for 2 years during the experimental work with mercurochrome in obstetrics. It is evident that a very small amount of the mercurochrome remains in the vagina and there are parts of the birth canal which remain free from the drug. In Figure 3 when the syringe is used properly, it will be noted that the solution distends the vault of the vagina, is undoubtedly forced into the cervix if open, and the entire vaginal mucosa comes in contact with the drug. If the labia are not held closely together or if the instillation is done when the cervix is fully dilated and retracted past the presenting part, it is evident that it is impossible to instill the vagina properly. This is the reason why we insist that the instillation be started as near the beginning of labor as possible. Two small sponges, held one on either labium, will keep the gloved hand from slipping and as the syringe is withdrawn and the excess fluid starts to escape, it may be collected in the syringe by releasing the bulb and moving the syringe from side to side. Any fluid not taken up by the syringe is absorbed by the sponges. If the labia are not held closely together around the

TABLE VII.—MORBIDITY BY MONTHS 1925 THROUGH 1929

	Deliveries	Morbidity	Per cent
January	707	58	8.2
February	659	63	9.5
March	737	74	10.04
April	737	57	7.7
May	790	49	6.2
June	728	54	7.4
July	822	64	7.7
August	813	47	5.7
September	778	65	8.3
October	782	53	6.7
November	773	63	8.02
December	703	38	5.4

TABLE VIII.—ETIOLOGY OF MORBIDITY FOR THE YEARS 1928–1930

Total deliveries	5102
Uncorrected morbidity	5.6
Corrected morbidity	3.01
Due to delivery	
Lochiometra	50
Endometritis	2
Parametritis	3
Sapræmia	17
Retained membranes	2
Retained placenta	2
Subinvolution	2
Infected perineum	9
Phlebitis	1
Bacteriæmia	1
Pelvic abscess	2
Salpingitis	1
Thrombophlebitis	1
Unclassified	22
No note	27
Examination negative	12
Not due to delivery	
Respiratory	42
Breast	40
Intestinal	5
Cholecystitis	2
Pyelitis	35
Otitis	1
Rheumatism	2
Genito-urinary	2
Pyonephrosis	2
Encephalitis	1
Parotitis	1

syringe, the barrel of the syringe tends to keep the mercurochrome in the vagina, but even in spite of this, a large part of the fluid will escape without ballooning the vaginal vault. We have had considerable difficulty in getting our internes to carry out this part of the technique.

A thick pad under the patient will absorb the spill from vagina and saves staining the bed linen.

Care of the perineum during labor. The perineum should be thoroughly cleansed, three moist sterile sponges being used, and the spraying and instillation should be repeated every 12 hours. It is very important to keep all the dried blood and mucus from collecting on the perineum. This should be removed every 2 or 3 hours during active labor.

Vaginal examination. The perineum is thoroughly cleansed with three or more moist sponges, the sponge being discarded as soon as it comes in contact with the anal region. The perineum should be sprayed. The labia are separated with the gloved hand, and the introitus is sprayed. The first two fingers are inserted into the vagina, the pelvic floor is depressed, and 2 drams of mercurochrome is put into the vagina from a small asepto syringe. (If the mercurochrome solution is used carelessly a large amount may be wasted. The cost of the mercurochrome should not be more than 10 or 20 cents per patient.) The separating and withdrawing of the fingers will allow the mercurochrome to reach the upper part of the vagina. When a vaginal examination is made at the time of admission, the mercurochrome need not be used in the vagina, but immediately following the vaginal examination, the vagina should be instilled as described above.

Preparation for delivery. The perineum and surrounding area should be cleansed with three or more moist, sterile sponges, all dried blood and mucus being removed, then dried with a sterile towel, and sprayed with a solution of 4 per cent aqueous, alcohol, acetone mercurochrome. The spraying should be done systematically, beginning over the pubes and moving the atomizer back and forth across the field as the bulb is pressed, until the whole area is covered. *Never use the acetone solution for the instillations.* After the pelvic floor is depressed, 2 drams of the aqueous solution are put into the vagina. If a forceps is to be applied, an induction to be done, or if considerable time is consumed in the delivery, more mercurochrome should be used in the vagina. If the perineum becomes soiled with fæces, it should be cleansed with a moist sponge, more mercurochrome instilled, or the perineum sprayed with mercurochrome. After delivery, if there is any laceration of the pelvic floor, or if an episiotomy wound is to be sutured, the blood should be cleared away and mercurochrome should be put into the wound before it is sutured.

Induction of labor. At the time of the induction, which should not be less than 1 hour after the original instillation, the patient is prepared as for delivery. The vagina is filled with a 4 per cent solution of mercurochrome. The cervix is located and the bag is passed through the cervix and filled with a weak solution of mercurochrome. After the stem has been tied, a hand sponge saturated with mercurochrome is inserted into the vagina. If the membranes have been ruptured for any considerable time or if the uterine cavity is considered potentially infected, a small catheter should be inserted into the uterus when the bag is introduced. After the bag is filled, 3 ounces of a 1 per cent solution of mercurochrome may be injected into the uterus through the catheter. The bag may remain in the uterus for 24 hours without danger of infection.

Cæsarean section. On admission to the hospital the patient should be prepared and instilled as in

"Preparation on Admission." This is very important if a cæsarean section is anticipated, even though the patient may not be in labor. If labor is prolonged, the instillations should be repeated every 12 hours. If the membranes are ruptured and the patient is considered potentially infected on admission, before it is endeavored to do a cæsarean section, a catheter should be passed into the uterus above the presenting part and 3 ounces of a 1 per cent solution of mercurochrome instilled into the uterine cavity. Following the cæsarean section and immediately upon the removal of the placenta, before the uterine cavity fills with blood, 1 ounce of a 4 per cent solution of mercurochrome is poured into the uterine cavity and allowed to drain out through the cervix and vagina. The mercurochrome solution should not be allowed to flow into the abdominal cavity. Any spill should be absorbed by sponges. When the fascia of the abdominal wall is closed, the wound is swabbed with mercurochrome, care being taken that no excess solution is left in the wound. A uterine pack saturated with mercurochrome may be left in the uterine cavity.[1]

Postpartum care. The perineum is sprayed at least once daily with a 4 per cent aqueous solution of mercurochrome. If there is a bad laceration of the perineum, a previous vaginitis, or if the patient is a poor obstetric risk, the vagina should be instilled daily with 2 drams of a 4 per cent solution of mercurochrome. This may be done with a small vaginal syringe, similar to the one illustrated in Figure 2, or by means of a catheter attached to a small syringe. This procedure has been made routine by Dr. Goodall, of Canada, and Dr. Hagstrom, of the Methodist Episcopal Hospital, Brooklyn, New York.

Dilatation and curettage including abortions and miscarriages. When the patient is admitted to the hospital, the perineum should be prepared and the vagina instilled as in "Preparation on Admission." This should be done at least 1 hour before the operation. The preparation at the time of the operation should be as in "Preparation for Delivery." All clots and blood should be removed, the vagina being left as dry as possible. The vagina is then filled with mercurochrome. The cervix is exposed and swabbed out with a stick sponge or narrow strip of packing saturated with mercurochrome. A strip of packing also saturated with mercurochrome is passed into the uterus. A small asepto syringe, containing 2 drams of mercurochrome, may now be used to fill the uterine cavity with mercurochrome. Care should be taken when instilling the uterus. If the uterine cavity is small, I have used a 2 cubic centimeter Luer syringe filled with mercurochrome and feel that this is sufficient. It is possible to force the mercurochrome through the tubes as was done in one case in which I did a laparotomy, following a dilatation and curettage. If there is a rupture of the uterus, the mercurochrome may escape into the peritoneal cavity. This is not desirable and may cause some discomfort following the operation. The uterus may again be instilled following the operation, as mentioned before, or, if it is decided to pack the uterus or vagina, gauze saturated with mercurochrome may be used. This packing may be left in for 24 or 48 hours.

[1]This procedure is endorsed by Dr. Polak, who writes as follows: "Cæsarean section: When the membranes have been ruptured and vaginal examinations made, we take a large piece of gauze, approximately 2 yards square, soaked in a 4 per cent solution of mercurochrome, from which the excess has been squeezed out, and place it in the fundus of the uterus after the placenta has been delivered and as we are sewing up the uterine wound; this stimulates the uterus to contraction, and the gauze is expelled in the course of 24 or 48 hours."

SUMMARY

1. All previous reports on the morbidity following the mercurochrome technique have included the cases delivered during the experimental stage.

2. The morbidity in 5,076 vaginal deliveries during the development of the mercurochrome technique was 8.9 per cent and with 5,102 cases with the present technique, it was 5.6 per cent. This is compared with 2,072 cases before the use of mercurochrome with morbidity 12.4 per cent.

3. A history of the development and an outline of the mercurochrome technique, year by year, is given with the resulting morbidity.

4. The average days' morbidity per patient was 0.40 before the use of mercurochrome; 0.46 during the experimental stage; and with the present directions carefully followed out, 0.26.

5. Without mercurochrome 1 patient in every 345 had a morbidity of over 20 days. In the experimental stage it was 1 in 390, while in the latest group, it was 1 in 728.

6. The season of the year has very little to do with morbidity when thorough vaginal antisepsis is carried out.

7. The corrected morbidity for the last 5,102 cases was 3.01 per cent.

CONCLUSIONS

A 6 year study of vaginal antisepsis in obstetrics at the Methodist Episcopal Hospital and the resulting morbidity following over 10,000 deliveries with the mercurochrome technique, seems to have proved that it is possible to protect almost every mother from the ravages of puerperal infection.

NOTE—Up to the present there have been 8,077 deliveries with the present mercurochrome technique, with but a single death from puerperal sepsis following the vaginal delivery of a viable child. The following is an analysis since August 1, 1930:

	Cases	Morbidity per cent
Total deliveries	2798	6.8
Cæsarean sections	103	47.5
Deliveries less cæsareans	2695	5.2
Corrected morbidity	....	2.5
Average days' morbidity per patient		0.20
Longest period of morbidity		15 days
Number of maternal deaths		4
Deaths from sepsis		0

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CARCINOMA OF THE TONSIL

A REVIEW OF ONE HUNDRED TWENTY-TWO HISTOLOGICALLY PROVED CASES TREATED 1921 TO 1928, INCLUSIVE

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From the Service of Dr. Douglas Quick, Memorial Hospital

CARCINOMA of the tonsil is not as infrequent as is generally believed. By the term tonsil is meant both the tonsil and tonsillar pillar, because the disease has usually so far progressed when first seen that the site of origin is not certain.

Pack and Le Fevre in their survey of 19,129 admissions to the Memorial Hospital between January, 1917, and January, 1929, found that 1.93 per cent were suffering from carcinoma of the tonsil. These lesions constituted 2.23 per cent of the malignant tumors included in the survey. Of the intra-oral group of tumors, carcinoma of the tonsil accounted for 9.64 per cent, more than ten times the incidence of lymphosarcoma, which was 0.83 per cent of the intra-oral tumors. In the oral group of carcinomata, the frequency of tonsillar cancer was exceeded only by those of the tongue, lip, and larynx, the percentage in the order named being 20.59, 15.39, and 12.17.

Because of the rôle the tonsils play in the infectious processes, we are apt to regard them merely as collections of lymphoid tissue. A brief review of the histology of the tonsil will show that epithelium constitutes a relatively large surface in the tonsillar region.

The lymphoid tissue of the tonsil is covered by the tunica mucosa, which consists of the tunica propria and the membrana propria. This epithelium is stratified epithelium of many layers, with flattened cells on the free surface and columnar cells beneath. The stratified epithelium serves as a lining to a varying number of macroscopic depressions, usually 10 to 20, which are the crypts. These are irregularly tubular and are sometimes branched. This epithelial covering of the tonsil with its many infoldings, together with the convex tonsillar pillar, constitute a relatively large area of epithelium though limited to a small region.

The exact etiology of carcinoma of the tonsil is unknown. If the theory of mal-development in the embryonic stage has any relation to the production of cancer, then it can be especially true of the tonsillar lesions, for in this region there is a complication association, in the embryo, of the hypoblastic and epiblastic tissues, pouching in to form the oral cavity.

The location of the tonsils is such that they are constantly subjected to irritation, by the constant motion of the jaws in speaking and eating, by thermal irritation consequent upon very hot foods, by smoking, and by the bacterial infections, which are more or less constantly present in the tonsillar crypts.

It is thought by some that cancer occurs only in tissue undergoing retrograde changes. Tonsils serve their greatest function in early life, and then usually show signs of progressive atrophy. Any one, or any combination, of these factors may be considered an exciting agent of tonsillar carcinoma.

There is no pathognomonic symptom of carcinoma of the tonsil. The most common symptom is pain, especially on swallowing. But this dis-

comfort accompanies so many oral lesions that it invites a thorough oral examination, even though the patient is only a child. Carcinoma of the tonsil has occurred in children under 5 years of age. At times the patient complains of an irritation or soreness of the throat, and occasionally of pain referred to the side of the head. Irritation and soreness indicate that the primary lesion is not widespread over the contiguous regions. If the pain referred to the side of the head is a prominent symptom, it will be found that the lesion infiltrates deeply and that there is considerable extension into the base of the tongue, or there is an obvious mass in the upper cervical region, which has infiltrated around the upper branches of the cervical nerves.

A painless mass in the upper cervical region, or overlying the carotid bulb, may be a metastasis from an apparently insignificant cancer of the tonsil. This manifestation is commonly associated with a lesion of the transitional cell type. It is not infrequent to have the patient apply at the hospital for treatment after such a mass has been incised by a physician, who, after a casual glance into the mouth, has opened an "abscess," only to drain blood. This procedure tends to spread metastases, and at times permits the growth to fungate through the wound.

The diagnosis of a cancer of the tonsil is not always simple. A microscopic section is sometimes necessary for a final decision. A classical description of a carcinoma of the tonsil is impossible; the sites of origin differ, the extent varies, there are different degrees of ulceration, bulk, infiltration, and especially a variable amount of infection, which alters the appearance.

The more usual type of tonsillar lesion might be described as an ulcerated growth of variable size, involving the tonsil and tonsillar pillar, commonly the anterior. The edges of the ulcer are elevated, indurated, irregular. The induration may extend into the tongue, in which case there is a fissure between the tonsillar pillar and the tongue. The floor of the mouth and the mucosa of the cheek are the next most likely regions to be involved. The ulcer may present the appearance of a clean, granular surface, or a sloughy, infected crater, or the intermediate stages.

The radiosensitive, non-ulcerating type of cancer may be confused with lymphosarcoma; and the cervical metastases from a very small but radiosensitive carcinoma of the tonsil, may be simulated by cervical Hodgkin's disease. The distribution of the adenopathy is helpful in differentiating carcinoma from Hodgkin's disease; in the latter, the adenopathy is usually general. But the lymph node enlargement is of little value in differentiating tonsillar cancer from lymphosarcoma. In either case the tonsil may be enlarged and a mass may be felt in the cervical region. A radiosensitive tonsillar carcinoma often lacks the characteristic induration which is frequently considered necessary for a diagnosis of cancer.

Assistance in the differential diagnosis of carcinoma and lues, or tuberculous ulcer, may be had by the Wassermann reaction, and X-ray films of the chest. But it must be borne in mind that either lues or tuberculosis, or both, may be coexistent with carcinoma. Diagnosis of carcinoma of the tonsil must never be made on clinical examination alone; a histological study of a section is necessary in every case, primarily for verification of clinical diagnosis, but also to classify the growth according to gradation of malignancy.

The region of the tonsillar pillar is not a rare site for an adenoid cystic carcinoma. This lesion can often be distinguished clinically by its lack of ulceration, and its consistence, which is that of encapsulated fluid under tension.

It is not necessary to mention the rarer types of tonsillar neoplasms, because the diagnosis necessarily depends on biopsy.

In the literature on tumors of the tonsil (and these are reports of one, or at most, a few cases), tonsillar sarcoma is often mentioned and described. The more common description is a non-ulcerated tumor, with the appearance of encapsulation. The mode of metastases is said to be the same as in carcinoma, and this extension occurs early. These so called sarcomata are undoubtedly carcinomata, of the transitional cell type, or the lympho-epithelioma, as described by Schmencke, Regaud, and Ewing. Sarcoma of the tonsil, other than lymphosarcoma which belongs to the lymphoma group, is rare. Pack and Le Fevre did not find a case recorded in their study of 19,129 cases admitted to the Memorial Hospital.

The surgical approach in the care of carcinoma of the tonsil has been most inadequate. Cheever, of Boston, was the first in this country to describe the surgical treatment by a lateral pharyngotomy. This was in 1870. Matthews (1912) did not have a cure in 22 cases. His treatment was tonsillectomy and cautery, or excision of the growth by a lateral pharyngotomy. This latter procedure was attended by a very high mortality; 4 operative deaths in 35 reported cases; 13 others died within 6 months. Only 4 patients lived beyond a 2 year period. Jacobson (1901) reported one patient free of disease 11 years after tonsillectomy and cautery. Bloodgood, in one of the latest texts of surgery, states that he has not cured a case of

carcinoma of the tonsil. Following the destruction of the primary growth by the cautery or diathermy, the patients have died of metastases.

This brief résumé should be sufficient to compel anyone to classify carcinoma of the tonsil in any stage as primarily surgically inoperable.

CLINICAL STUDY

It is my purpose to report a clinical study of 122 histologically proved cases of carcinoma of the tonsil admitted to the Memorial Hospital between the years 1921 and 1928, inclusive.

The average age in this series was 55.4 years. The two extremes were 78 and 21 years. The patient 78 years old had a papillary growth, grade I, radioresistant. The youngest patient, 21 years of age, had a transitional cell type carcinoma, grade III, radiosensitive. In the latter case there was bilateral cervical involvement. For 9 months patient had noticed a swelling in his neck, which had been incised before he applied to the hospital for treatment.

It is of no practical importance whether the lesion is on the right or the left side. In this series, the right and left sides were almost equally involved: 52 lesions were on the right side, and 69 were on the left side.

Since carcinoma of the tonsil is never a primarily operable disease, it cannot be classified into operable and inoperable, or into early, borderline, and advanced, which is a surgical nomenclature and refers to surgical technique rather than to prognosis. In this communication, the carcinomata will be classified as early or advanced. By early is meant that the lesion involves only one, or at most, two contiguous regions, that is, tonsil and tonsillar pillar, or tonsillar pillar and adjacent tongue, etc. If three regions are involved, or wide extension into an adjacent region is found, then the case is advanced. It is not uncommon to have a cancer of the tonsil infiltrate the base of the tongue to a point near the opposite tonsil. In one patient in this series a growth of the tonsil had so deeply infiltrated the mucosa of the cheek that there was marked trismus, due to infiltration of the masseter muscle by the neoplasm.

Of the 122 cases studied, only 33 were classified as early, while 88 were advanced. Two patients had had a tonsillectomy before admission, so were classified as early, and one chart was deficient in its description, although the diagnosis was verified both historically and histologically.

Several cases were classified as early though cervical metastases were present. The reason for this classification is to emphasize the fact that insignificant primary lesions can give rise to metastases.

If only those cases were considered early, in which the primary lesion was limited to two regions, and at the same time showed no neck involvement, the total would be 14, or 11.5 per cent.

The gradations, as determined by the histological study, of the sections of the 117 classified cases, were as follows:

	Cases	Per cent
Grade I	10	8.5
Grade II	88	75.2
Grade III	8	6.9
Transitional	11	9.4

In 4 cases the section was so small it could not be graded, and one was a mixed grade II and transitional cell carcinoma.

These figures at first sight would seem to be at slight variance with those given by Ewing in his classification of 200 tonsillar lesions. But if the grade III group, which has lost its squamous characteristics, is combined with the transitional cell group, and the single case of mixed type added, then, within a small percentage, the figures agree with his total of 72 per cent squamous and 18 per cent lympho-epithelioma and transitional cell carcinoma.

There is much to be learned by the histological study of very early lesions of the tonsil. If the tissue removed by tonsillectomy were carefully sectioned and microscopically studied, we would eventually learn much about the beginning of carcinoma primary in the tonsil itself. The exact location and the mode of origin are not now known.

The treatment of carcinoma of the tonsil is usually a combination of external and interstitial radiation. Each side of the neck is subjected to high voltage X-ray, the beam including both the primary lesion and the lymph drainage areas. Immediately following these exposures, the radium pack is directed over the primary lesion and the adjacent cervical region. These radiations are given in full erythema dosage. This amount of radiation delivers 150 per cent to 180 per cent of a skin erythema dosage to the tonsillar growth. With the histological gradation and the clinical response to the external radiation as a basis for judging the radiosensitivity, a varying amount of radon is then implanted into the neoplasm, depending on its size and the configuration.

The number of patients who apply for treatment with cervical metastases already definitely established, and often in the advanced stage, calls for a more vigorous education of the public re-

TABLE I.—COMPOSITE CHART OF ALL TONSILLAR CARCINOMA CASES TREATED FROM 1921 TO 1928, INCLUSIVE

Year and no. cases	Average age and extremes	Location	Early or advanced	Pathological gradation				Condition of cervical region on admission			External radiation	Nodes developed	Local recurrence	Ligation	Local neck dissection	Complete neck dissection	Recurrence in neck	Hæmorrhage	Average duration of slough	Average interval of those dying	No. well over 5 years
				I	II	III	Trans.	No nodes	Operable nodes	Inoperable nodes											
1921 15	55.5 41-63	6 right 9 left	6 early 9 advanced	2	12	0	1	8	5	2	Heavy 1 Minimum 14	3	5	6	4	0	1	6	5 mos.	7 mos.	3
1922 10	58.5 42-78	5 right 5 left	3 early 7 advanced	1 1 not specified.	8	0	0	7	3	0	Minimum 6 None 4	5	4	7	4	0	2	1	5 mos.	16 mos.	1*
1923 15	54.7 42-68	7 right 8 left	5 early 10 advanced	0 1 not specified.	12	0	2	8	2	4	Minimum 8 None 7	2	3	3	2	1	2	1	6 mos.	14 mos.	4
1924 9	51.1 45-68	5 right 4 left	4 early 5 advanced	0	4	1	4	3	5	2	Heavy 7 Minimum 2	3	3	8	2	2	1	4	5½ mos.	26 mos.	2
Total 49		23 right 26 left	18 early 31 advanced	3	36	1	7	26	15	8	Heavy 8 Minimum 30 None 11	13	15 30%	24 50%	12	3	6	12 24%	5⅓ mos.		10 of 49 20.4%
1925 17	56.4 44-67	9 right 8 left	6 early 11 advanced	2	13	2	0	5	7	5	Heavy 13 Minimum 4	2	4	3	3	1	3	0	4 mos.	15 mos.	
1926 18	58 36-78	12 right 6 left	6 early 12 advanced	3 2 not specified.	10	2	1	7	7	4	Heavy 15 Minimum 1 None 2	2	6	6	3	1	2	5	2 mos.	15 mos.	
1927 13	51.4 37-66	3 right 10 left	1 early 11 advanced †	2 1 mixed, II transitional	7	3	0	3	3	7	Heavy 13	1	2	2	2	0	1	3	2 mos.	12 mos.	
1928 25	56.9 21-71	6 right 19 left	2 early 23 advanced	0	22	0	3	10	5	10	Heavy 25	2	6	2	4 (2 inflammatory)	3	5	3	1¼ mos.	14 mos.	
Total 73		30 right 43 left	16 early 57 advanced	7	52	7	4	25	22	26	Heavy 66 Minimum 5 None 2	7	18 24.5%	13 18%	12	5	11	11 15%	2 mos. ‡		
Total 1921–1928 122 cases		53 right 69 left	34 early 88 advanced	10	88	8	11	51	37	34	Heavy 74 Minimum 35 None 13	20	33	37	24	8	17	23			

*Died of tuberculosis, not cancer.
†One not specified, though proved historically and histologically.
‡Average increased because of the second implantation required in several cases.

TABLE II.—1921 TO 1924. NO NODES ON ADMISSION

	Primary	Grade	Recurrence	Development of nodes	Where	When	Operation	Result
1921 1	Early	II	0	0	0	0	0	Well 8¼ yrs.
2	Advanced	II	Tonsil 2 mos.	0	0	0	0	Died 5 mos.
3	Early	II	Floor of mouth 6 mos.	+	Right upper deep cervical	3 mos.	Carotid dissection	Died 10 mos. Not free of disease. Cardiorenal.
4	Advanced	II	0	0	0	0	0	Died 2 mos. Hæmorrhage.
5	Advanced	II	Tonsil 3 mos.	0	0	0	0	Died 8 mos. Primary uncontrolled.
6	Advanced	II	0	+	Carotid bulb	1 mo.	0	Died 2 mos.
7	Advanced	I	Tonsil 1 mo. Tonsil 5 mos.	+	Angle of jaw	6 mos. direct extension	0	Died 17 mos.
8	Advanced	II	0	0	0	0	0	Well 9 yrs.
1922 9	Advanced	II	0	+	Submaxillary	2 mos.	0	Died 4 mos.
10	Advanced	II	0	+	Upper deep cervical	5 mos.	Carotid dissection	Died 17 mos.
11	Advanced	II	Anterior pillar 5 mos.	0	0	0		Died 27 mos.
12	Early	II	Tongue 1 mo.	+	Submaxillary	1 mo.	Submaxillary dissection	Died 18 mos.
13	Early	II	Tongue 9 mos.	+	Scar of ligation	9 mos.	0	Died 10 mos.
14	Early	?	0	+	Upper deep cervical	7 mos.	0	Died 33 mos.
15	Early	I	0	0	0	0	0	Died 34 mos. Bronchopneumonia.
1923 16	Early	II	Tongue 5 mos.	+	Upper deep cervical	1 mo.	0	Died 18 mos.
17	Advanced	II	Tongue time ?	+	Left submaxillary posterior	14 mos.	0	Died 16 mos.
18	Advanced	II	0	0	0	0	0	Died 2 mos. Hæmorrhage.
19	Early	II	0	0	0	0	0	Lost 15 mos. No evidence of disease.
20	Advanced	II	0	0	0	0	0	Died 8 mos. Pneumonia.
21	Advanced	II	0	0	0	0	0	Well 7⅓ yrs.
22	Advanced	II	0	0	0	0	0	Well 7⅔ yrs.
23	Advanced	Carcinoma but insufficient to grade		0	0	0	0	Well 7½ yrs.
1924 24	Early	II	Soft palate 4 mos.	+	Upper deep cervical	6 mos.	0 inoperable	Died 14 mos.
25	Early	II	Epiglottis 2 yrs.	+	Upper deep cervical	3 yrs.	0 inoperable	Died 40 mos.
26	Early	III	Base of tongue	+	Upper and lower cervical	3 mos.	+	Died 27 mos.

garding the dangers of any oral lesion. This education might better be directed toward the medical profession, many members of which are often satisfied to give an alkaline mouth wash or simply paint the affected tonsil with silver nitrate. The patient is then told to return at a future date. Many of the advanced cases are the result of such a procedure.

Of the 122 cases studied in this series, 51, or 41.8 per cent, had no palpable metastases when admitted for treatment. Thirty-seven, or 30 per cent, had surgically operable cervical metastases, and 34, or 28 per cent, presented themselves when the disease had so far progressed in the cervical region that surgical dissection would have been impossible.

TABLE III.—1925 TO 1928. NO NODES ON ADMISSION

	Primary	Grade	Recurrence	Development of nodes	Where	When	Operation	Result
1925 1	Advanced	II	o	o	o	o	o	Well 55 mos.
2	Early	II	o	o	o	o	o	Well 56 mos.
3	Advanced	II	Alveolar ridge 8 mos.	+	Upper deep cervical	4 mos.	Dissection incomplete	Died 12 mos.
4	Early	II	Base of tongue 6 mos.	+	Submaxillary region	2 mos.	o	Died 20 mos.
5	Advanced	II	o	o	o	o	o	Well 56 mos.
1926 6	Advanced	II	Base of tongue 1 mo.	+	Upper deep cervical	1 mo.	Complete dissection	Well 44 mos.
7	Advanced	II	Base of tongue 4 mos.—also 2 yrs.—5 mos.	o	o	o	o	Lost 29 mos. Going bad.
8	Early	Grade II and transit.	Tonsil 3 mos. Base of tongue 7 mos.	+	Upper deep cervical. Posterior triangle	3 yrs. 3 yrs.-5 mos.	o	Died 48 mos.
9	Advanced	II	o	o	o	o	o	Died 13 mos. cause ?
10	Advanced	I	Never controlled	o	o	o	o	Died 3½ mos.
11	Early	II	o locally, œsophagus 4 mos.	o	o	o	o	Died 4 mos. Carcinoma of œsophagus and tuberculosis.
1927 12	Advanced	Insufficient to grade but carcinoma	o	o	o	o	o	Well 54 mos.
13	Advanced	II	o	+	o	o	o	Died 3 mos.
14	Advanced	II	o	o	o	o	o	Died 17 mos. Not carcinoma—mental case.
15	Advanced	II	o	o	o	o	o	Well 36 mos.
1928 16	Advanced	II	Tongue 4 mos. Tongue 9 mos.	Inflammatory o	o	o	Dissection	Lost 1 yr., 10 mos. No evidence of disease.
17	Advanced	II	o	o	o	o	o	Well 29 mos.
18	Advanced	II	Tongue 3 mos.	Inflammatory o	o	o	Dissection	Died—œsophagus carcinoma. 20 mos.
19	Advanced	II	o	+	Upper deep cervical	6 mos.	Inoperable	Died 16 mos.
20	Advanced	II	Tonsil and pharyngeal wall 5 mos.	Inflammatory o	o	o	Dissection	Died 13 mos.
21	Early	II	o	Inflammatory o	o	o	Dissection	Well 28 mos.
22	Advanced	II	o	o	o	o	o	Died 7 mos. Hæmorrhage.
23	Early	II	o	o	o	o	o	Well 29 mos.
24	Advanced	Transit.	o	o	o	o	o	Well 30 mos.
25	Advanced	II	Alveolar ridge	+	Upper deep cervical	5 mos.	o	Died 32 mos.

Ninety-one patients were admitted to the hospital with cervical metastases or later developed them. The location of these metastases was in the upper deep cervical group in 58 instances, 63.7 per cent. The node overlying the carotid bulb was first affected 12 times, and the node in the posterior submaxillary region 6 times, or 13.2 and 6.6 per cent, respectively. The opposite side to the primary lesion was first palpably involved in 3 instances, 3.3 per cent. This same number of times the whole chain of nodes on the side of the primary lesion was palpably invaded. Both

TABLE IV.—1921 TO 1924. OPERABLE NODES ON ADMISSION

	Primary	Grade	Recurrence	Location of nodes	Operation	Recurrence in neck	Result
1921 1	Early	II	0	Deep cervical	Ligature and bare seeds	0	Died 3 mos. hæmorrhage
2	Early	II	0	Upper deep cervical	Carotid dissection	0	Died 6 mos. hæmorrhage
3	Early	II	0	Upper deep cervical	Carotid dissection	0	Lost 5 yrs. No evidence of disease
4	Advanced	I	0	Upper deep cervical	Carotid dissection	0	Died 6 mos. hæmorrhage
5	Advanced	III Transit.	0	Upper deep cervical	Carotid dissection	0	Died 10 mos.
1922 6	Advanced	II	Tongue 3 mos.	Upper deep cervical	Excision of node and bare seeds	0	Died 9 mos.
7	Advanced	II	0	Upper deep cervical	Incomplete dissection	0	Died 5 yrs. Tuberculosis; no carcinoma
8	Advanced	II	0	Upper deep cervical	Excision at ligation	0	Died 2 mos. Pneumonia at age 81
1923 9	Advanced	II	Primary not controlled	Upper deep cervical	Incomplete dissection	0	Died 3 mos.
10	Advanced	Transit.	0	Upper deep cervical	0	0	Well 7 yrs.
1924 11	Early	III Transit.	0	Upper deep cervical	Dissection incomplete and bare seeds		Died 6 mos.
12	Advanced	III Transit.	0 Recurred in left upper quadrant 1 yr. 5 mos.	Upper deep cervical	Dissection complete and bare seeds	0	Died 19 mos.
13	Advanced	Transit.	0	Carotid region	Dissection incomplete	0	Well 5 5/6 yrs.
14	Early	II	0	Deep cervical	Dissection complete	0	Well 7 3/4 yrs.
15	Advanced	II	Primary not controlled	Upper deep cervical	Incomplete dissection	0	Died 5 mos.

sides of the neck were involved 8 times, 9.5 per cent. It is interesting to note that following a ligation of the carotid artery and its branches, a neoplastic deposit was found in the scar once.

The treatment of the cervical region in carcinoma of the tonsil is the same as in cancer in other locations of the oral cavity. Only a very brief résumé will be made in this treatise. The conservative treatment of the cervical region has been maintained. On admission to the hospital service, the lymph drainage areas of the neck are irradiated with radium or X-ray or both. The site of the primary lesion is included in the beam of rays. This procedure is followed whether or not there is definite manifestation of disease in the neck. At the completion of this external radiation, gold tubes of radon are implanted into the primary growth. If there are no cervical metastases present at the time of admission, the patient is observed carefully and at frequent intervals, with special attention to the neck. If operable cervical metastases develop, or if they are present at the time of admission, a neck dissection is done when the skin reaction from the external radiation has subsided and the primary lesion is about healed. The time for this to obtain is usually 3 to 6 weeks.

Cervical nodes to be classified as operable must be limited to the same side of the neck as the primary lesion, the carcinoma must not have infiltrated the capsule, and the gradation of the lesion must be in the more fully differentiated class histologically.

When a dissection is indicated, a full and complete operation is done, including the anterior and posterior triangles, with resection of the sternomastoid muscle and the jugular vein. If the primary growth is of a very radiosensitive type, radical surgery of the nodes is not undertaken; dependence is placed upon the surgical exposure of the node and radon in full dosage implanted in gold tubes. In the more advanced cases, the interstitial radiation is buried through the skin without incision. This latter method is used in advanced inoperable cases, if the type of cell of the neoplasm is radiosensitive or radioresistant.

Only in rare instances can "cross" metastases or bilateral metastases be considered operable.

In evaluating the efficacy of external radiation, it will be more enlightening to compare the group

TABLE V.—1925 TO 1928. OPERABLE NODES ON ADMISSION

	Primary	Grade	Recurrence	Location of nodes	Operation	Recurrence	Result
1925 1	Early	II	o	Upper deep cervical	o	o	Died 5 mos.
2	Advanced	II	Soft palate	Upper deep cervical	o	o	Died 6 mos.
3	Early	III	o	Upper deep cervical	o	Opposite side 11 mos.	Died 3 yrs. 8 mos.
4	Advanced	II	Anterior pillar 7 mos.	Upper deep cervical and submaxillary	o Refused	o	Lost 1 yr. 3 mos. Doing badly
5	Advanced	II	o	Upper deep cervical	Dissection complete	Under angle of jaw 2 yrs. 6 mos.	Died 2 yrs. 9 mos.
6	Advanced	II	Lobe, tongue soft palate	Carotid bulb	Carotid dissection	o	Died 1 yr. 2 mos.
7	Advanced	I	o	Post submaxillary region	o	o	Died 4 mos.
1926 8	Advanced	I	o	Upper deep cervical	o External radiation; patient 78 yrs.	o	Died 6 mos.
9	Early	II	Tongue 4 mos. uncontrolled	Carotid bulb	Dissection complete	Submaxillary region 5 mos.	Died 23 mos.
10	Early	II	Anterior pillar and soft palate 9 mos.	Upper deep cervical	Dissection incomplete	o	Died 2 yrs. 11 mos. of carcinoma of stomach
11	Advanced	III	o	Upper deep cervical	o	o	Died 6 mos.
12	Advanced	Transit.	o	Carotid bulb	External radiation and gold tubes at exposure	o	Lost 9 mos. Not free of disease
13	Advanced	II	Base of tongue 8 mos.	Carotid bulb	Dissection incomplete	Scar 6 mos.	Died 18 mos.
14	Advanced	II	Not controlled. Only external radiation	Upper deep cervical	o Patient 74 yrs.	o	Died 2 mos.
1927 15	Advanced	II	o	Submaxillary	Submaxillary dissection	o	Died 9 mos. alcoholic
16	Advanced	I	Soft palate 6 mos.	Upper deep cervical and (2) posterior submaxillary	Upper neck dissection	Opposite neck 11 mos.	Died 1 yr. 5 mos.
17	Advanced	II	o	Carotid	Anterior dissection	o	Well 2 yrs. 5 mos.
1928 18	Advanced	II	Uvula 1½ mos.	Upper deep cervical	Complete dissection	o	Died 1 yr. 3 mos. Cause ?
19	Advanced	II	o	Carotid bulb	Incomplete dissection 6-23-28. Submaxillary 1-25-29	o	Well 1 yr. 5 mos.
20	Advanced	II	o	Upper deep cervical	Complete dissection	Incision 6 mos.	Died 9 mos.
21	Advanced	II	Uvula 2 mos.	Upper deep cervical	Complete dissection	o	Died 15 mos.
22	Advanced	II	o	Upper deep cervical	Complete dissection	Upper neck 3 mos.	Died 9 mos.

of cases of 1921–1924, inclusive, and the group 1925–1928, inclusive. It was about 1925 that the external radiation dosage was increased, and later the 4 gram radium element pack was added to the hospital equipment.

Of the 26 cases admitted without palpable nodes between 1921 and 1924, inclusive, 13 patients or one-half developed cervical metastases. But many of these died of uncontrolled primary, intercurrent disease or hæmorrhage, within too short a time to make a worth while estimate. A better evaluation, even though made on a smaller number of cases, can be had by including only those patients who lived 2 or more years. There were 10 such cases. Of these 10, only 3, or 33 per cent, developed cervical metastases.

From 1925 to 1928 there were 25 patients admitted without cervical adenopathy. Eighteen, or 72 per cent, did not develop malignant nodes of the neck. Of the 13 patients living 2 or more years, 3 have had involvement of the cervical nodes, 23 per cent.

Comparing the two periods, it is found that from 1921 to 1924, inclusive, 50 per cent of all cases remained free of regional metastases, and 66 per cent of those living 2 years or more had

TABLE VI.—1921 TO 1924. INOPERABLE NODES ON ADMISSION

	Primary	Grade	Location of mass	Treatment	Result
1921 1	Advanced	II	Confluent mass upper deep cervical	Partial removal and bare seeds	Lost 10 mos.
2	Early	II	Upper deep cervical	External radiation	Died 3 mos.
1923 3	Advanced	II	Upper deep cervical infiltrating sternomastoid	External radiation and bare seeds	Died 17 mos.
4	Advanced	II	Nodes both sides	External radiation and bare seeds, both sides	Died 17 mos.
5	Early	III Transit.	Upper deep cervical and submaxillary	External radiation	Died 6 mos.
6	Advanced	II	Whole chain of nodes	External radiation; bare seeds	Died 1 mo.
1924 7	Advanced	III Transit.	Upper deep cervical same side. Carotid on opposite side	Bare seeds	Died 6 mos.
8	Early	II	Upper deep cervical and submaxillary	External radiation and bare seeds at exposure	Well 5 yrs. 2 mos.

no evidence of cervical involvement. During the period 1925 to 1928, inclusive, the respective percentages were 72 per cent and 77 per cent.

There is considerable percentage difference in the results in the two periods. More adequate external radiation during the second period was undoubtedly a factor, but due consideration must be given to the use of gold tubes after the beginning of 1925. Gold tube implants of radon have been much more efficacious in the destruction of the primary growth than the glass seeds. While there is disease present, danger of metastases persists. Recurrence is a manifestation of latent disease.

The location and frequency of local recurrences were as follows: tongue, 16 times; tonsil, 7; soft palate, 6; pharyngeal wall, 2; and floor of mouth and mucosa of the cheek, once each.

During the period 1921 to 1924 there were 15 local recurrences, 30 per cent, whereas in the period 1925 to 1928 there were 18 local recurrences, 24 per cent. This decrease in the percentage of local recurrences is primarily due to the use of gold tubes, this type of implant being first used at the beginning of 1925; the bare seeds were in use previous to that time.

The reason for the decrease in recurrences will be obvious if the quality of the radiation from the two types of implants is considered. If the amount of radiation transmitted by the glass seed is taken as 100 per cent, then the relative amounts of β-rays and γ-rays are 96.5 per cent and 3.5 per cent, respectively. With the 0.3 millimeter of gold filter, the β-radiation is 8.8 per cent and the γ-radiation is 91.2 per cent, again assuming that the transmitted radiation is 100 per cent. The total radiation transmitted by the gold tube is only 3.16 per cent of that transmitted by the glass seed. The 0.3 millimeter of gold screens out practically all the soft, necrosing β-rays, whose sphere of activity is limited to a region with a diameter less than a centimeter, but which, due to their quantity, produce total destruction of tissue in proximity to their source. The usual amounts of radon in glass seeds and gold tubes are 1 millicurie and 2 millicuries, respectively. In the comparison of these two implants, it is found that the glass seed of 1 millicurie produces five times the volume tissue necrosis as the gold tube of 2 millicuries. In other words, twice the amount of γ-radiation may be had from gold implants, with one-fifth the volume necrosis produced by glass seeds. Hence more equal distribution of radiation is delivered throughout the tumor bearing area.

The complete necrosis around the implant was a great difficulty in the care of patients treated with glass seeds of radon. The slough became an excellent culture medium for bacteria, and the infection added to the slough. The average duration of the presence of the slough following the use of glass seeds was 5.4 months. This average was reduced by using gold tubes to 2 months. Many of the patients having had gold tube implantation into the tonsillar region had no slough at any time. The average was brought up to 2 months either by the presence of infection due to poor oral hygiene or by the necessity for further treatment of a persistent portion of the disease or a recurrence. These conditions are essentially the same; only the time interval suggests the distinction. The second implantation of radon, even in gold filtered tubes, nearly always causes a secondary necrosis.

TABLE VII.—1925 TO 1928. INOPERABLE NODES ON ADMISSION

	Primary	Grade	Location of mass	Treatment	Result
1925 1	Advanced	I	Angle of jaw and over carotid bulb	External radiation and gold tubes	Lost 8 mos.; doing badly
2	Advanced	II	Opposite side of neck	Gold tubes followed by dissection	Died 3 mos.
3	Early	III	Both sides of neck	Gold tubes	Died 9 mos.
4	Advanced	II	Upper deep cervical	External radiation	Died 18 mos.
5	Advanced	II	Upper deep cervical	External radiation	Died 7 mos.
1926 6	Advanced	?	Deep cervical infiltrating muscle and fluctuant	External radiation and gold tubes	Died 4 mos.
7	Advanced	III	Both sides of neck	External radiation, gold tubes each side	Died 19 mos.
8	Early	II	Carotid region 5 x 10 cm.	External radiation	Died 5 mos.
9	Advanced	III	Upper neck region	External radiation	Died 4 mos.
1927 10	Advanced	II	Deep cervical both sides	External radiation	Died 10 mos.
11	Advanced	?	Upper cervical 8 x 6 cm.	External radiation	Died 8 mos.
12	Advanced	III	Both sides	External radiation	Died 3 mos. with abdominal metastases
13	Advanced	I	Same side—whole chain	External radiation	Died 2 yrs. 2 mos.
14	o Tonsil elsewhere	III lectomy	Upper deep cervical infiltrating sterno-mastoid 5 x 6 cm.	External radiation; 28,000 at 6	Died 1 yr. 6 mos.
15	Early	III	Upper deep cervical	External radiation and gold tubes	Well 2 yrs. 7 mos.
16	Early	II	Upper deep cervical	Gold tubes and external radiation	Died 4 mos.
1928 17	Advanced	II	Upper deep cervical	External radiation and gold tubes	Died 11 mos.; hæmorrhage
18	Advanced	II	Carotid region	External radiation and gold tubes	Died 1 yr. 4 mos. Died 2nd carcinoma of pharyngeal wall
19	Advanced	II	Opposite upper deep cervical	External radiation	Died 3 mos.; hæmorrhage
20	o Cervical recurrence	II	Opposite carotid region	External radiation and gold tubes	Died 6 mos.
21	Advanced Both tonsils	II	Right angle of jaw. Lt. carotid region	External radiation and gold tubes	Died 7 mos.
22	Advanced	II	Deep cervical 11 x 5 cm.	External radiation	Died 2 mos.
23	Advanced	III	Deep cervical 8 x 8 cm.	External radiation and gold tubes	Died 1 yr. 10 mos.
24	Advanced	III Transit.	Both sides	External radiation and gold tubes	Died 1 yr. 10 mos.
25	Advanced	II	Deep cervical	External radiation	Died 7 mos.
26	Advanced	II	Upper deep cervical	External radiation	Died 6 mos.

Consequent upon the necrosis and the separation of the slough, hæmorrhage occurs. The region of the tonsil has an abundant blood supply, and the inaccessibility precludes the possibility of tamponage, which in this region obstructs the breathing. Ligation of the external, lingual, and facial arteries is the only procedure at all adequate to control the hæmorrhage; this suffices only if the bleeding is arterial, it is less efficacious if the hæmorrhage is venous.

During the period that the glass seeds were in use, ligation was done in 50 per cent of the cases, and there were 24 per cent of the patients who had hæmorrhage. Following the advent of the gold tubes, a ligation was done of 18 per cent of the patients, and bleeding occurred in 15 per cent. These figures do not adequately describe the improvement in the technique, because the severity of the bleeding has been markedly decreased.

Sufficient time has not elapsed to study the 5 year results in those cases treated with gold tubes.[1] This portion of the statistical study includes only the tonsillar carcinomata treated previous to 1925.

[1]The data for this paper were collected in 1929.

and with bare seeds. There were 49 cases in this group and 10 survived the 5 year period, a percentage of 20.4 per cent. One patient was lost just at the 5 year interval. He was free of disease, and if included in the 5 year cases, the percentage would be 22.4 per cent.

If the patients are grouped according to the condition of the cervical nodes, 5, or 38.4 per cent, of the 13 cases with no nodes throughout their period of observation, are well. No patient is well who entered the hospital without nodes, but later developed them after admission. There were 13 such cases. Of those 15 patients who had palpable metastases but in an operable stage on entrance to the hospital, 4 patients, or 26.6 per cent, survived the 5 year period. In the inoperable group, 1, or 12.5 per cent, is clinically free of disease at the end of 5 years and 2 months. This patient had a grade II lesion, with the upper deep cervical group of nodes involved. Following the regression of the primary lesion and dissection of the anterior triangles of the neck, with removal of the sternomastoid muscle and the jugular vein, metastases appeared in the posterior triangle of the neck. Since the primary lesion had completely regressed, this manifestation of disease was undoubtedly present, but not discernible, at the time of operation; therefore, it is classed in the inoperable group because of the wide dissemination. However, this classification may be questioned. The nodes in the posterior triangle were implanted with radon in glass seeds.

Whichever way this case is classified, the 20.4 per cent clinically free of disease at the end of 5 years remains the same.

TABLE VIII.—CASES WITH NO NODES ON ADMISSION, EXTERNAL RADIATION GIVEN AND THE SUBSEQUENT DEVELOPMENTS

Primary lesion	Grade	Radiation	Developed nodes	When	Result
1921					
Early	II	Minimum	o		Well 8½ yrs.
Advanced	II	Minimum	o		Died 5 mos.
?	II	Minimum	+	3 mos.	Died 10 mos.
Early	II	Minimum	o		Died 2 mos.
Advanced	II	Minimum	o		Died 8 mos.*
Advanced	I	Minimum	+	6 mos.	Died 17 mos.
Advanced	II	Minimum	+	1 mo.	Died 2 mos.
Advanced	II	Minimum	o		Well 9 yrs.
1922					
Advanced	II	Minimum	+	2 mos.	Died 4 mos.
Advanced	II	Minimum	+	5 mos.	Died 17 mos.
Advanced	II	None	o		Died 27 mos.
Early	?	None	+	7 mos.	Died 33 mos.
Early	II	Minimum	+	1 mo.	Died 18 mos.
Early	II	None	+	9 mos.	Died 10 mos.
Advanced	I	Minimum	o		Died 34 mos.†
1923					
Early	II	Good	+	1 mo.	Died 18 mos.
Advanced	II	Minimum	+	14 mos.	Died 16 mos.
Advanced	II	Minimum	o		Died 2 mos.‡
Early	II	Minimum	o		Lost 15 mos.§
Advanced	II	Minimum	o		Died 8 mos.†
Advanced	II	Minimum	o		Well 7⅔ yrs.
Advanced	II	Minimum	o		Well 7⅓ yrs.
Advanced	?	Minimum	o		Well 7¼ yrs.
1924					
Early	II	Minimum	+	36 mos.	Died 40 mos.**
Advanced	II	Fair	+	6 mos.	Died 14 mos.
Early	III	Fair	+	3 mos.	Died 27 mos.
1925					
Early	II	Good	o		Well 55 mos.
Early	II	Fair	o		Well 56 mos.
Advanced	II	Fair	+	4 mos.	Died 12 mos.
Early	II	Minimum	+	2 mos.	Died 20 mos.
Advanced	II	Good	o		Well 56 mos.
1926					
Advanced	II	Good	+	1 mo.	Well 44 mos.
Advanced	II	Fair	o		Lost 29 mos. Going bad
Early	II	Fair	+	36 mos.	Died 48 mos.
Advanced	II	Fair	o	Lost	Died 13 mos.
Advanced	I	Fair	o		Died 3 mos.
Early	II	Good	o		Died 4 mos.
Advanced	?	Good	o		Well 54 mos.***
1927					
Advanced	II	Fair	+	2 mos.	Died 3 mos.
Advanced	II	Fair	o		Died 17 mos.****
Advanced	II	Fair	o		Died 36 mos.
1928					
Advanced	II	Fair	o		Well 29 mos.
Advanced	II	Good	o		Died 20 mos.
Advanced	II	Fair	+	6 mos.	Died 16 mos.
Advanced	II	Fair	o		Died 13 mos.
Early	II	Good	o		Well 28 mos.
Advanced	II	Fair	o		Died 7 mos.
Early	II	Fair	o		Well 29 mos.
Advanced	Transit.	Good	o		Well 30 mos.
Advanced	II	Good	+	5 mos.	Died 32 mos.
Advanced	II	Good	o		Lost 22 mos.§

*Primary uncontrolled.
**Local recurrence 26 mos.
***Carcinoma of the œsophagus.
****Died, not carcinoma.
†Pneumonia.
‡Hæmorrhage.
§No evidence of disease.

SUMMARY

Carcinoma of the tonsil is not a rare disease; it comprises 2.23 per cent of all the malignant tumors admitted to the Memorial Hospital, and 9.64 per cent of the intra-oral malignant tumors.

Surgery alone is totally inadequate to cope with this disease.

Most carcinomata of the tonsil are advanced when first seen in the hospital clinic.

The size of the primary growth has no relation to the stage of the disease as judged by the metastases.

More adequate external radiation and the use of gold filtered tubes of radon have made radiation therapy more efficacious.

(The grade II type of carcinoma was the preponderant lesion—88, or 75.2 per cent.)

Of the 122 cases in this series, only 51, or 41.8 per cent, were admitted without cervical metastases. Only those patients treated up to 1925 can be included in the 5 year group: Of these 49 cases—13 had no cervical nodes throughout and 5, or 38.4 per cent, are well; 13 developed metastases after admission and none is well; 15

TABLE IX.—DURATION OF LIFE AFTER ADMISSION

1921–1924 inc.	No. of cases	0–6 mos.	6 mos.–1 yr.	1–2 yrs.	2–3 yrs.	3–4 yrs.	4–5 yrs.	5 yrs. over
No nodes throughout	13	4—30% (3 hæmorrhage. 1 uncontrolled primary)	2—15%	1—7%	2—15%	—	—	4—30%
Developing nodes	13	2—15%	1—7%	6—46%	3—23%	1—8%		—
Operable nodes	15	4—27%	5—33%	1—7%	—	—	—	5—33%
Inoperable nodes	8	1—12%	4—50%	2—25%	—	—	—	1—12%
TOTAL	49	11—22%	12—24%	10—20%	5—10%	1—2%	—	10—20.4%
1925–1928 inc.	No. of cases	0–6 mos.	6 mos.–1 yr.	1–2 yrs.	2–3 yrs.	3–4 yrs.	4–5 yrs.	5 yrs.
No nodes throughout	18*	3—16%	1—6%	4—23%	1—6%	—	2—12%	—
Developing nodes	7†	1—14%	—	3—43%	1—14%	—	1—14%	—
Operable nodes	22‡	4—18%	8—36%	6—27%	1—4%	1—4%	—	—
Inoperable nodes	26§	5—19%	10—38%	8—31%	2—8%	—	—	—
TOTAL	73	13—18%	19—26%	21—29%	5—7%	1—2%	3—4%	—

*7 cases no evidence: 4 3/5, 3 3/4, 2 1/2, 2, 1 3/4, 1 3/4, 1 1/4 years.
†1 case no evidence: 3 2/3 years.
‡2 cases no evidence: 2 1/2 and 1 1/2 years.
§1 case no evidence: 2 1/2 years (grade III external radiation and gold tubes).

had operable nodes on admission and 4, or 26.6 per cent, are well; and 8 had inoperable nodes on admission and 1, or 12.5 per cent, is well. In the group treated 5 or more years ago, 10 patients are now clinically free of disease, from 5 to 9 years, a percentage of 20.4.

Carcinoma of the tonsil should be treated by radiation therapy, or by radiation and surgery of certain metastases; not by surgery alone.

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THYROIDECTOMY FOR THYROTOXICOSIS[1]

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IT is a matter of every day experience that the timid or inadequate removal of the thyroid commonly gives but limited relief from symptoms and only partial control of the rate of metabolism. It is equally a matter of every day clinical experience and laboratory demonstration that total or subtotal removal of the normal or pathological thyroid results in varying degrees of hypothyroidism, closely related to the extent of thyroid removal.

In my own experience, the extent of the thyroidectomy has determined not only the degree of immediate relief, but the permanence of that relief. I have been able consistently to substitute hypothyroidism for hyperthyroidism in a series of cases, the data of which I will present to you. In the great majority of the patients the hypothyroidism ultimately gave way to normal thyroid activity with what I judged to be the development of a compensatory hyperplasia. Hypothyroidism, however, persisted in a sufficient number of these patients to present the only source of doubt as to the advisability of this radical procedure. That some failed to return to normal function and that those whose function returned to normal did not develop by a progressive increase in the thyroid function to above normal, that is, to relapse, I have attributed to sheer inability of the small amount of thyroid that I leave to develop such a relapse. The persistence of hypothyroidism in the human subject has its counterpart in the failure of adequate compensatory regeneration in the experimental animal in which the amount of residual thyroid is sufficiently reduced.

In comparing results of the radical operation which I propose with less radical procedures regarded by many as satisfactory, I have repeatedly stated that objective data based on observations for which the reporter can personally vouch, are alone of value.

The diagnosis of the patient's postoperative condition is just as important and requires the same type of study as the original diagnosis. Neither can be accomplished by correspondence. It requires no unusual clinical experience to learn that the subjective feeling of perfect well-being on the part of the patient may be associated with the most obvious objective evidence of persistent thyrotoxicosis. No answers to letters of inquiry can have any diagnostic value that justifies their consideration.

As a justification for the very radical operation advocated in this paper, I wish to present in abstract the recent studies of two series of cases previously published. For this purpose data on the basal metabolic rate alone are presented because I believe they form the simplest and most accurate objective data available for comparative studies. It must be clearly understood, however, that not only were these metabolism studies made under my personal supervision, or the supervision of competent recognized internists, but that the patients were also personally examined under the same conditions. That is, the diagnosis of the postoperative condition was based upon exactly the same type of study as that on which the original pre-operative diagnosis was made. No data on the basal metabolic rate or reports on the condition of the patients made by their family physicians or by outlying laboratories or hospitals are included.

The statistical material is limited to patients who have been adequately studied, and whose basal metabolic rate generally was above +30. Plus thirty was arbitrarily chosen to safeguard against the inclusion of questionable border-line cases. Patients who were clinically toxic, but whose metabolic rate was not raised, or whose clinical data were insufficient, were separately classified and not included in this study. There were no deaths among these, so the mortality data are not favorably affected by excluding them. A certain number had received iodine preparation before coming under observation; where the clinical diagnosis was obvious, these were included. Relatively few were included with typical thyrotoxicosis of mild grade with consistently raised metabolic rate but not reaching +30. In all, 14.8 per cent were below +30.

The first series (1) concerned 112 consecutive patients of whom a personal postoperative study of 100 could be made. This series was studied in June, 1926, and published in March, 1927. At that time 99 of this 100 had normal or subnormal rates of metabolism, 5 after a secondary operation. Seventy-six of these patients have since been followed; 55, for over 5 years. Two hundred and thirty-eight additional metabolic rate readings have been made upon them. But one has been

[1]Presented before Sectional meeting of the American College of Surgeons, Oakland, California, April 23-26, 1931.

found to be toxic. She was a child of 13 when operated upon, hence a less radical operation was done. She had but a single metabolic rate reading of +6 at the time of the original publication, then disappeared for 6 years. She now is mildly toxic (+28) and has palpable thyroid masses.

The second series was of 500 cases published in July, 1929 (2). Of these, 447 were available for postoperative study at the time of publication. Four hundred and forty-three of these had a basal metabolic rate of +15 or below; 3 of the 4 obviously were not toxic at the time, their metabolic rate being accounted for in the paper as published. None of this series had required a secondary operation.

Three hundred and fifty-four of these patients have since been studied, 655 additional metabolic rate readings being made to date. Of these, the patient who was toxic at the time of publication was subsequently re-operated upon. One had a single metabolic rate at the time of publication, of +3. Subsequently she had two raised readings and was clinically toxic, though her metabolic rate is now +10. One had an associated hæmolytic jaundice at the time of operation. Her metabolic rate had not reached normal on the eighth postoperative day when she passed from under observation. She returned 4 months later in an acute exacerbation of her jaundice, during which she died. Two metabolic rate readings taken during this exacerbation, were +20 and +23. Fourteen of the 43 patients on whom we had no data at the time of publication returned for further study. Of these, one who had regarded himself as well for 2½ years, was obviously toxic.

Thus, of 600 patients subjected to ultraradical thyroidectomy the data on which have been published, 559 have had some postoperative study. Of these 430 have been subjected to further studies including 900 additional metabolic rate readings. But 2 of those reported normal at the time of publication have since become toxic, each after a single normal basal metabolic rate. One of these 2 was the 13 year old child mentioned whose operation was planned to be relatively conservative because of her youth. In not a single patient in this series whose basal metabolic rate was normal on two or more tests has there been a relapse of thyrotoxicosis with a rise of metabolic rate.

A further study of 500 additional patients subjected to thyroidectomy subsequent to these two series shows but a single patient, mentioned below, who, after three normal basal metabolic rate readings, showed a definite rise with evidence of thyrotoxicosis. This single possible exception in a series of 1,100 cases was a patient whose metabolism tests were made at long intervals, with practically no medical supervision, during which he may very well have had periods of thyrotoxicosis. It is conceded that thyrotoxic patients not subjected to operation may have periods of quiescence; certainly, thyroidectomy, ever so incomplete, may not prevent this. The very great number of conservative thyroidectomies that are followed by permanent benefit show how wide a latitude is permitted the surgeon.

To determine the nature of what is commonly described as a "relapse," a study was made of a series of 50 patients presenting themselves for secondary operation following failure of a previous thyroidectomy. Thirty-two had their first operation elsewhere, 18 by me. Most of these patients reported themselves as having been "well" for various periods of time following their original operation, ranging from 8 months to 13 years. Forty-one of these patients had had no adequate medical supervision, and no basal metabolic rate studies during their period of seeming well-being. Eight of these patients had been adequately studied clinically and with repeated metabolic rate observations. All 8 of these were found to have been consistently toxic throughout. Some had occasional normal or low metabolic rates especially while taking iodine—none had consistently normal rates at any time. These were all obvious failures and not relapses.

One patient, mentioned, had shown three normal readings and may possibly be recorded as a case of true relapse after operative cure, but was not under adequate supervision, and cannot be accurately classified. Thus, of 50 unselected apparent relapses, but one incompletely studied patient may have had a true relapse; while 8 patients adequately studied proved to be cases of inadequate surgery.

Mortality. The ultraradical thyroidectomy in one sitting is planned not merely to lessen the incidence of immediate failure and late relapse of thyrotoxicosis, but to lower mortality. The basic idea (3) is that not enough pathological thyroid is left to permit a serious postoperative thyroid crisis. According to this conception, it is precisely the most toxic patients, the poorest risk patients, who require the most radical operation, and that at one sitting.

There was one death in each of the two series, a total of 2 deaths in 612 cases, a patient mortality of 0.3+ per cent.

In a study of results in older patients, we found that in a consecutive series of 1,060 patients of whom 200 were over 50 years of age, there were 6

deaths among the 200, and 3 deaths in 860 consecutive cases under 50 years of age, a total mortality of 0.85 per cent. The patients were not picked, their ages ranged to 76 years; and no patient whose consent could be obtained was rejected. Obviously, the mortality would have been further lowered by less rigid exclusion of clinically toxic patients on their basal metabolic rate findings.

THE ESSENTIAL FEATURES OF THE OPERATION

1. *Pre-operative.* Iodine is given in large doses, well diluted, over a period of 2 to 4 weeks, longer in severe cases. Clinical gain rather than metabolic rate response determines the duration. My experience with small doses of iodine has been unsatisfactory. Medicinal preparation other than iodine and sedatives is left entirely to the internist. Except for special indications, patients are ambulatory and not hospitalized during this period, though long hours of rest and high caloric feeding is enforced.

2. The thyroidectomy is completed in one sitting. There are no preliminary ligations or lobectomies. The purpose is to leave so little thyroid behind that a postoperative reaction cannot develop.

3. The upper poles are so exposed as to permit the division of the superior thyroid arteries between clamps. These are the only vessels divided between clamps during the entire operation. No other vessel requires it. The poles themselves are never ligated—no part is ever left behind.

4. With the pole drawn downward the fascial tissue forming a sort of suspensory ligamentous structure supporting the upper border of the isthmus and containing a single small artery is made tense. A curved forceps is gently insinuated under it, toward the median line, a clamp is applied and it is divided.

5. The isthmus is divided and the parts retracted laterally by means of sharp retractors hooked into the tissue, thus everting the lobe. Usually but one artery, or none, crosses the isthmus and needs clamping—the retraction prevents venous bleeding. The large veins crossing over its surface may be disregarded if kept under gentle tension. The size of the isthmus has little bearing on its vascularity.

6. Sharp and dull dissection from the trachea permit the lobes to be further everted. A vascular tree can often be made out in the inner surface, and grasped with forceps; otherwise one, or in large lobes, two forceps are pushed into the thyroid tissue close to but not impinging on the trachea, and the lobe further dissected off and everted.

7. One or two forceps are now pushed into the lower pole close to the capsule, one or two into the outer aspect of the lobe parallel with the capsule, and one on the posterior aspect of the lobe near the upper pole, and the lobe is removed. Both lobes are treated alike. In the great majority of hyperplastic thyroids, a single forceps is placed on each superior thyroid artery and six or less, usually four or five, in each lobe. Often additional forceps or a suture are further applied after removal of the lobes—seldom more than two or three additional ligatures in all.

The larger nodular goiters commonly require more forceps merely because of anatomical distortion. If the forceps could be as correctly placed as in the symmetrical goiters, very few additional forceps would be required.

Care is taken to save as complete, but as thin a layer of posterior and mesial capsule as possible, for the protection of the nerves and parathyroids.

The thyroid tissue remaining is spread over a very thin layer. One can roughly estimate its dimensions in cubic centimeters and record the result in grams. I have gradually been reducing the amount so that it is now less than 2 grams, but I am inclined to leave more, probably 2 to 3 grams, because of the persistence of hypothyroidism in too many of my patients.

Since little thyroid is left behind, little or no postoperative iodine is required. I have recently reduced the postoperative use of iodine to a moderate dose the day of operation, little or none thereafter unless some reaction suggests that more than the estimated amount of thyroid has been left behind. Since most of these patients are temporarily hypothyroid, desiccated thyroid is started about 4 weeks after operation and given in varying doses, guided by the clinical symptoms and metabolism findings as long as may be necessary, usually several months to a year or more.

Immediate effect on the basal metabolic rate. Following this radical thyroidectomy the metabolic rate after an abrupt rise during the first 24 hours, falls rapidly to or below normal. In a series of cases studied by the Massachusetts General Hospital Goiter Group, the metabolic rate was found to reach normal in an average of 10 days. In my study, 21 consecutive patients were subjected to daily postoperative basal metabolic rate studies. In 20 of these the rate reached normal on or before the sixth day, averaging 4+ days. Of these patients, 9 were operated upon on a Monday, and all 9 had normal rates on Saturday, 7 of the 9 on Friday, an average of 3.55 days. Twelve were operated upon on Tuesday; no readings were made on Sunday, and 11 of the 12 were normal on

Monday, the twelfth on Tuesday; an average of 4.66 days.

This drop in basal metabolic rate was undoubtedly substantially influenced by the postoperative administration of iodine but is more rapid than that which occurs in the normal individual on the withdrawal of thyroid that has been administered in excessive doses.

SUMMARY

The proposition which I wish to present is that:

Adequate thyroidectomy as I have described it, reduces the basal metabolic rate to normal, with disappearance of all evidence of intoxication, within a few days.

Relief from hyperthyroidism is permanent.

Completing the operation in one sitting prevents severe postoperative reaction and reduces the mortality to a minimum.

A failure to accomplish our purpose is never due to failure of the operation, but of the operator.

What is loosely spoken of as a "relapse" is practically always a residual hyperthyroidism due to inadequate removal of the thyroid.

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ACUTE AND CHRONIC INFECTIONS OF THE PAROTID GLAND

Treatment by Dilatation of Stenson's Duct

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INFLAMMATION of the parotid gland has been known since the time of the ancients. "In the first book of the Prorrhetika, Hippocrates makes frequent reference to inflammatory swellings in the vicinity of the ears, which he describes as occurring in certain febrile and cachectic conditions. In indicating the topography of these, he says that they lie beside the ears. Before the days of Pliny these merely descriptive Hippocratic phrases had crystallized into a proper name—the name of a disease. In several passages, as for example, xx, 1, 15, 21; xxviii, 7 to 11; xxxv, 17, Pliny gives the names of remedies which are used for the disease parotides. Galen, in a like manner, makes a number of references to this disease." This "author specifically states that the parotides are abscesses beside the ears, called by some castores. Celsus (vi, 16) gives a brief account of this disease; and Paulus Ægineta describes at length the method of treatment which he considers most satisfactory (iii, 23)."

An analysis of the voluminous literature on infections of the parotid has been briefly and clearly done by Jennings in the *American Journal of Surgery* for June, 1930, and more elaborately in an admirable paper by Rankin and Palmer in the December, 1930, issue of the *Annals of Surgery*. We have, therefore, deleted the historical phase from this report. Briefly, there are two theories of the etiology of this disease generally accepted; hæmatogenous infection (the pyæmic theory) and infection from the mouth through Stenson's duct (the ascending theory). No attempt has been made to separate the cases reported; those reported by one author all apparently proving one theory and those by another as conclusively proving the opposite. Jennings suggests that in many instances both factors are present. When we turn to the treatment, however, we realize that Galen's concept of inflammations of the parotid as being abscesses is still generally accepted. All sorts of treatment including hot and cold applications, gum chewing, acid mouth washes, mercurochrome intravenously, heliotherapy, etc. are advocated in various articles, the writer very often ending his paper with a description of the incision which he uses in incising the gland. Rankin and Palmer offer what appears to be the most successful treatment to date—the use of radium. This treatment is undoubtedly of great value but unfortunately it is not at the immediate command of the average practitioner and surgeon.

All authors agree that as soon as suppuration can be definitely recognized, the gland should be incised. A certain percentage of these cases, however, will recover without being opened. Rankin and Palmer advise waiting until the process is well advanced and state that frequently drainage from Stenson's duct may occur, thus relieving the condition. This point will be elaborated upon in the discussion.

Case 1. On December 24, 1924, one of us (W.H.H.) saw a patient, a young married woman, age 27, complaining of a bilateral swelling in the parotid region, and of considerable pain. This condition had existed for about one year intermittently. There was no history of an infection immediately preceding the first appearance, her only recollection being that the face became tender and swollen. Heat was applied and after a few days the swelling subsided. This recurred many times during that year. Alpine light, gentle massage, ice caps, gum chewing, and other known therapeutic measures were tried without bettering the heat therapy. X-ray pictures taken during this time were negative for calculi. When first seen, the physical examination was negative, except for tender, hard swellings of both parotids. The mouths of Stenson's duct were red and œdematous. Pressure over the gland and along the duct was not productive. A small filiform was passed through the duct well back to angle of the jaw, its withdrawal being followed by cloudy secretion. Larger filiforms were then passed until the duct was well dilated. Massage of the gland and duct produced about a dram of cloudy secretion containing small shreds of pus. The same procedure was carried out on the other side with a similar result. Two cubic centimeters of 1 per cent mercurochrome solution was instilled into each gland. This procedure was repeated twice at weekly intervals. To date the patient has been free from symptoms.

This procedure was carried out in several cases during the next 3 years with relief of the symptoms. In a conversation with the late Dr. Joseph Sailer a question arose concerning infections of the parotid gland and one of us (W.H.H.) cited his experiences. Dr. Sailer seized upon the use of dilatation of the duct in infections of the parotid as a procedure which was rational and suggested that further work along this line be carried out. The co-authors were invited to assist in this problem in the fall of 1928. At this time the idea

of visualization of the gland by the use of a radio-opaque substance injected into the duct was evolved, without knowledge of the fact that this had been done previously. Sodium iodide was suggested, but this was rejected because of its irritating properties. Lipiodol was used in the studies which we will present.

As a control for the experiments which we hoped to carry out, it was deemed necessary that we obtain exact knowledge of the structure of the duct and gland from an anatomical and physiological standpoint. An attempt was made to dilate Stenson's duct in cadavers, inject the gland, and take X-ray pictures thereof. We were unable satisfactorily to get into the ducts without dissection which was impossible on our material, and which was not considered essential to our study. In the explanation of the following anatomical studies of normal glands in the living subject by the injection method, we have accepted the structure of the gland as described in standard textbooks of anatomy.

The parotid gland consists of a main and accessory gland which lies below and in front of the external ear, overlapping the upper part of the ramus of the mandible both within and without.

It is a compound racemose gland consisting of numerous lobes divided into lobules, each lobule of which is formed by the ramifications of a single duct, the branches terminating in dilated ends or alveoli on which the capillaries are distributed.

Stenson's duct is described as being 40 millimeters in length. It begins with numerous branches from the anterior part of the gland, crosses the masseter muscle and at the anterior border of this muscle turns nearly at right angles inward, passes through the corpus adiposum of the cheek, and pierces the buccinator, running between the mucous membrane and this muscle for a short distance and then opens upon the oral surface of the cheek by a small orifice opposite the second molar tooth. It receives the duct of the accessory portion while crossing the masseter muscle (Figs. 1 to 4).

The duct is dense, its walls being of considerable thickness. The canal is about the size of a crow's quill, but is greatly reduced in size at its oral opening. At this point let us add this statement from Gray's *Anatomy:* "The walls of Stenson's duct consist of a thick, external, fibrous coat, which contains contractile fibers, and of an internal coat lined with short columnar epithelium." It is analogous to, although not exactly the same as, the ureter. Like the ureter, it empties by peristalsis-like waves. Figure 5 shows this emptying clearly.

INSTRUMENTS FOR DILATATION

The instruments used by us to dilate Stenson's duct are very simple. Graded whalebone filiforms are inserted until the duct will allow the entrance of a 22 or 20 gauge needle. We have been using 4 inch needles with rounded points. Shorter ones will do, however. The larger bore needle is better for lipiodol injections. In one case, a No. 5 rat tail wax filiform was finally used and the patient at the present time passes it on himself. A 5 cubic centimeter hypodermic syringe was used both for irrigating and for injection of the lipiodol.

The cheek is grasped between the thumb and first finger of one hand, a piece of gauze being used to prevent slipping. The orifice of Stenson's duct is then located by the appearance of a drop of saliva after the buccal mucous membrane has been dried. If the orifice is not at once apparent, we blow air gently on the area with an air syringe, this having the double effect of drying the mucous membrane and of forcing apart the lips of the meatus. We experienced difficulty in finding the orifice only in our normal cases in the aged. Here the lack of teeth caused sagging of the cheek with a distortion of the normal landmarks. Once we have located the meatus, we insert the smallest filiform, passing it into the duct until it is stopped by the edge of the masseter muscle. At this point the cheek is stretched, thus straightening out the duct and enabling the probe to slide along until stopped by the narrowing of the canal. The distance the filiform may be passed varies with the patient, i.e., the size of the duct, and we must admit, with the operator. At first we experienced difficulty in entering the meatus and later, in passing the edge of the masseter. As we became more proficient, we entered more easily and more deeply; in three different ducts, we traversed a distance of 75 millimeters (almost 3 inches). This is like ureteral catheterization in which the beginner, having found the meatus, at first finds a stricture at this point, and later at various points along its course. As already stated, the duct is gradually dilated until it will admit the needle. This is then passed as far as possible although never as far as the filiform, and the saline or mercurochrome is instilled. The patient complains of a sense of fullness and some pain during the instillation. If done very slowly, not more than 2 or 3 cubic centimeters of the solution being used, the pain will not be unbearable. No anæsthetic solution was used and none is deemed necessary. No patient objected to further treatments when necessary. In making our plates, we inserted the needle to the edge of the masseter only and injected from 1 to 1.5 cubic centimeters

Fig. 1. Normal parotid, right side, showing ramifications of the ducts and terminal branches.

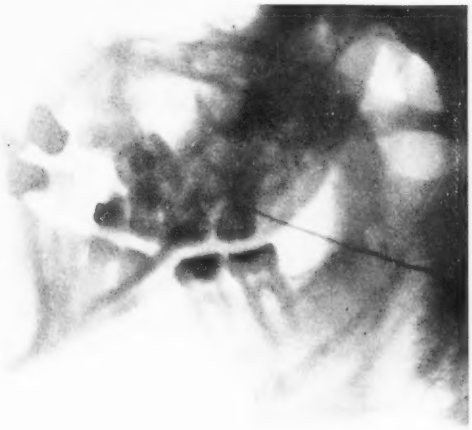

Fig. 2. Normal Stenson's duct, right side, showing its relation to adjacent structures.

of lipiodol. An immediate picture was taken with the needle containing the obturator in the canal, the patient holding this in place with his lips. Later plates were taken at 15 and 45 minutes with the needle removed. The only complaint during this procedure was of the taste of the lipiodol.

ADDITIONAL CASE REPORTS

CASE 2. M. B., female, aged 40 years. This patient was seen on February 2, 1929 on the tenth day following a cholecystectomy for acute cholecystitis with stones. The temperature which had been normal went to 103 degrees. Accompanying this, there was swelling and pain in the right parotid region. The duct orifice was pouting and thick yellowish mucoid material could be expressed. The duct was dilated and irrigated with saline solution three times that day and twice the following day. Two cubic centimeters of 2 per cent mercurochrome were instilled each time. There was an immediate relief from the pain and the swelling subsided in about 48 hours. Twenty-four hours later the left parotid swelled with the reappearance of the same symptoms. This duct was also dilated and irrigated and the procedure repeated 24 hours later. Relief was immediate. The swelling disappeared in the next 2 days. Recovery was otherwise uneventful. The patient

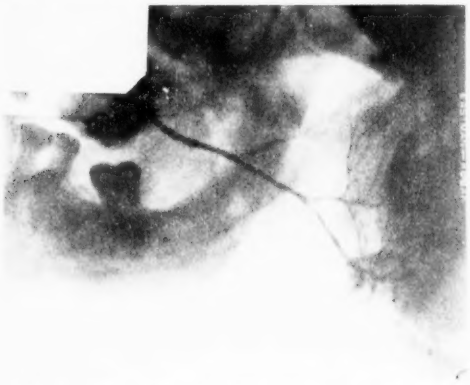

Fig. 3. Normal parotid, right side, showing accessory duct. Stenson's duct is distorted near its oral opening due to pressure of the needle which is still in place.

Fig. 4. Normal Stenson's duct seen from above. At the top of the plate is the gland end of the duct while at the bottom is the oral opening.

Fig. 5. Normal emptying of gland through Stenson's duct demonstrating peristaltic action.

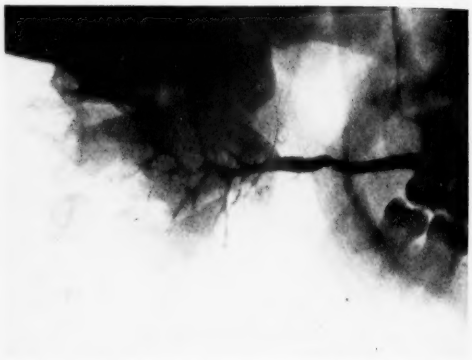

Fig. 6. Dilated and irregular Stenson's duct and duct branches within the gland following infection.

was discharged 3 weeks after admission completely relieved of all symptoms. Before discharge, a lipiodol injection picture was taken (Fig. 6).

CASE 3. L. J., aged 58 years, male. Following a one stage prostatectomy on April 5, 1929, the patient convalesced uneventfully until the tenth day. At this time the left parotid swelled, accompanied by pain and increase in temperature. The duct orifice was red and pouting and thick mucoid material could be expressed. The usual procedure was done with rapid relief and was repeated the following day. Recovery was uneventful.

CASE 4. E. G., aged 55 years. April 25, 1929. Both parotids became swollen in the course of an attack of grippe of 2 weeks' duration. The patient was very uncomfortable. He was seen 2 days after the onset of the swelling at which time the typical appearance of acute parotid inflammation was noted. His temperature was 101 degrees. Both ducts were draining profusely. The usual technique was done and the recovery was uneventful.

CASE 5. W. R. P., aged 60 years, female. The patient denied any acute illness but stated that about June 5, 1929, she noted a sudden swelling in front of the right ear. This was accompanied by consistent pain and with elevation of temperature to 99½ degrees. She was treated by her private physician for 1 week and then referred for surgical treatment. Examination on June 15, showed marked swelling and brawny œdema of the right parotid region. The area was red but no fluctuation could be noted. The buccal mucous membrane was œdematous. The swelling was so marked as to hide the orifice of Stenson's duct. This was found with great difficulty and only after careful drying of the mucous membrane. Massage of the cheek caused a few drops of apparently normal saliva to appear. The orifice would only admit the obturator of the needle used for instilling lipiodol. The duct was gradually dilated until a No. 22 gauge needle could be passed and a few drops of 2 per cent mercurochrome were instilled. This aggravated the condition immediately so that morphine was required for relief. It was decided to wait for fluctuation before incising, and therefore wet applications were used. Three days later the gland was opened and a large amount of pus was obtained. This was not cultured. The wound healed in about 2 weeks.

CASE 6. L. R. C., aged 48 years, physician, December, 1928. In 1924 the patient had a slight attack of grippe. Two weeks afterward the right parotid region suddenly became swollen and tender with extreme pain on eating and on moving the jaw. The symptoms subsided in a few days under local applications. Since then, the patient had had intermittent swelling of the parotid every few weeks. At this time, the pain was so intense that he was unable to work. This condition annoyed him to such an extent that he was willing to undergo any surgical treatment which was deemed necessary if relief could be assured. Treatment by the use of chewing gum, massage, alpine light, and various other procedures had given only temporary relief. When seen, the right parotid region was but slightly swollen, and no signs of acute inflammation could be noted. The orifice of the duct was prominent but not inflamed. On pressure, a small amount of apparently normal saliva was obtained. The duct was dilated and 2 per cent mercurochrome was instilled. The end of the larger filiforms showed a small amount of impacted whitish material. The patient returned at 6 week intervals for dilatation and irrigation. He stated that on the day following each treatment the gland was tender and painful but that in the interim, he was much better than he had been at any time previously. This continued until September 10, 1929, at which time a No. 5 rat tail filiform was passed and the patient was taught to use this upon himself. He was seen once during the winter of 1930, when he stated he was much improved On January 10, 1931, the patient stated that he had remained quite comfortable since the last treatment. At times the gland became tender but the pain was never intense and subsided in a day or so following dilatation by the filiform. Examination on this date showed no evidence of any swelling and no tenderness. The meatus appeared normal. Lipiodol pictures were taken on September 8, 1929, and September 10, 1930 (Figs. 7 and 8).

CASE 7. W. S., aged 21 years, male, seen March 10, 1920. One month previously the patient noted sudden swelling in the right parotid region following an attack of grippe. There was some pain on separating the jaws. The swelling was not large and subsided in a few days, with hot wet applications. For the past 2 weeks he had complained of pain on eating and he was therefore referred by his physi-

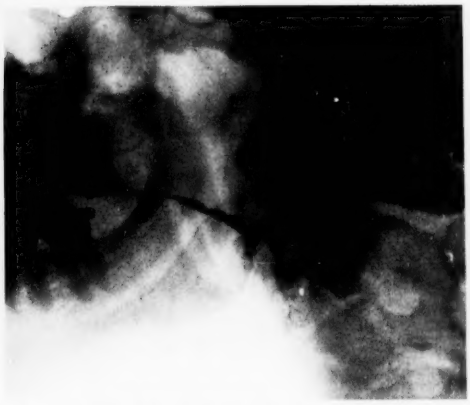

Fig. 7. First picture of Case 6. Note the pocket-like formations within the ducts of the gland and irregularity of Stenson's duct.

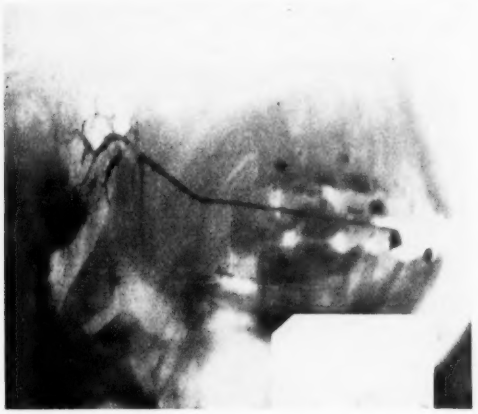

Fig. 8. Second roentgenogram of Case 6 taken about one year later. The pocket-like formations are less pronounced.

cian for treatment. Examination of the gland externally was negative. The duct orifice was normal. Filiforms were easily passed along the canal and a lipiodol picture was taken (Fig. 9). No further treatment was advised and the patient has not returned.

CASE 8. E. S., aged 10, was seen on April 20, 1929. Three weeks previously the patient had had a swelling of the left parotid. It was diagnosed as mumps and treated expectantly. The swelling gradually subsided in about a week. There was still, however, some fullness of the area and the patient complained of pain on eating. She was referred for treatment. Externally, there was a faint swelling over the gland which was slightly tender. The duct orifice was normal in appearance. Filiforms were passed easily and a lipiodol picture taken (Fig. 10). No further treatment was advised and the patient has not returned.

CASE 9. E. T. T., aged 75 years, female, seen November 22, 1929. About 1 year ago this patient suffered from a painful swelling in the left parotid region, following an attack of grippe. It subsided in a few days but had recurred frequently since. She stated that at no time was she severely ill with this condition. She had tried several types of treatment but could not say that they helped her. She stated that eating did not make it worse. The main symptom aside from the recognized pain was dryness of the mouth. She also complained of lack of appetite. Otherwise she felt normal. Physical examination was negative aside from slight swelling in both parotid regions, more marked on the left. Both glands were tender to palpation. The orifices of both ducts were red and pouting. Filiforms were passed into each canal without difficulty and pressure immediately following caused the appearance of a thick, mucilaginous, flaky secretion. Both ducts were irrigated with saline and a lipiodol picture was taken of the left. (Fig. 11.) On December 2, 1930, the patient stated that the pain was practically gone. The swelling was almost imperceptible at this time. The ducts were again dilated and irrigated. On February 7, 1930, the patient stated that she was entirely cured. No treatment was given. She reported by letter on April 6, 1930, that there had been no recurrence of the swelling and pain.

CASE 10. L. E., aged 30 years, female. Patient stated that about 6 weeks previously an ulcerated, right upper, molar tooth had been removed. Two weeks later the right side of her face became swollen and tender. This was accompanied by severe pain on any motion of the jaw. Acute symptoms gradually subsided under local applications but the area was still tender and painful. Physical examination when seen (January 10, 1930) was negative except for a marked tender swelling over the right parotid region. No signs of acute inflammation were noted. The orifice of the right duct was somewhat injected and pouting. The left ductal opening was normal. Both ducts were dilated. The secretion from the left was clear and apparently normal. That of the right was thick and contained white flakes. Both glands were irrigated with saline solution. A lipiodol picture was taken of the right gland (Fig. 12). On January 10, 1930, the patient stated that the pain was better. The swelling was markedly less. The right duct was again dilated and irrigated. On January 18, 1930, the right gland was normal in size and not tender. The patient stated that it felt perfectly normal. No treatment was given. She was last seen on July 7, 1930, at which time there had been no recurrence of the pain or swelling.

CASE 11. A. K., aged 69 years, female, was seen on June 3, 1930. On the eighth day of an attack of bronchopneumonia the patient developed bilateral swelling of the parotid. The crisis had occurred on the sixth day so that the temperature was 99½ degrees prior to this infection. The temperature rose to 103 at this time and was accompanied by extreme pain. Both meati were pouting and red and thick pus could be expressed from each duct by massage of the gland. The usual technique was carried out and about a half a dram of pus expressed from each duct. The patient experienced considerable relief almost immediately. The procedure was repeated daily for 5 days, although the swelling subsided in about 48 hours. There was no recurrence of the condition. The patient gradually failed however, and died of vascular failure after all parotid symptoms were gone.

CASE 12. R. M., aged 65 years, male, was seen on May 21, 1930. This patient was seen 10 days following a suprapubic cystotomy for vesical calculus. He had not rallied from the operation and had become progressively weaker. At the time seen, he was semiconscious. The day previous he had developed swelling of the right parotid

Fig. 9. An apparently normal gland.

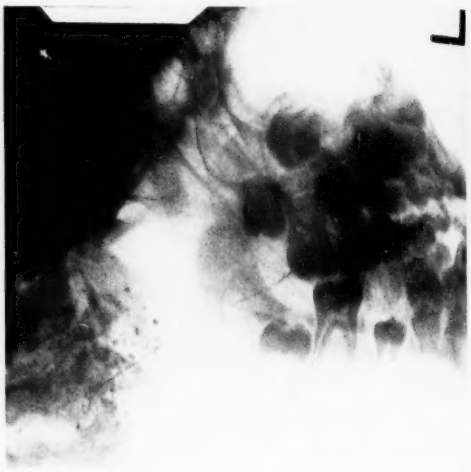

Fig. 10. Shot-like appearance of gland following mumps. Duct normal.

gland, with a rise of temperature. When seen his temperature was 103 degrees. The parotid region was markedly swollen. The mouth was very foul. The duct orifice was pouting and red, and thick purulent material could be expressed. The duct was dilated and irrigated with saline. This treatment caused no change in the temperature or the general condition of the patient. He gradually failed and died 12 hours later. At the time of his death there was no change in the appearance of the gland and the duct.

CASE 13. M. T., aged 40 years, female, was seen September 8, 1930. This patient was seen on the ninth day following an abdominal operation for ectopic pregnancy. Her convalescence had been uneventful up to this time when she suddenly developed swelling of both parotids with rise of temperature and severe pain. The duct openings were red and pouting and thick, purulent material could be expressed. She received dilatations daily for 5 days. Mercurochrome was also instilled the first three times. All parotid symptoms disappeared by the tenth day. The patient was discharged from the hospital 3 weeks after admission (Fig. 13).

CASE 14. A. M., aged 60 years, male, was seen November 12, 1930. Following a perineal prostatectomy under spinal anæsthesia, the patient became uræmic. He was kept alive by repeated intravenous and subcutaneous injections of saline, but was comatose at all times. On the third day the right parotid swelled. There was no temperature, however. Examination showed the usual appearance of acute inflammation. The duct was dilated and irrigated with saline. A large quantity of purulent material was expressed. This had no effect upon his general condition and he died 24 hours later.

CASE 15. (This patient was not seen by the authors.) On the third day following an appendectomy in a young woman, the left parotid became swollen. This was accompanied by rise of temperature and considerable pain. The duct was dilated and irrigated with saline once by an interne who had heard our preliminary paper. No further dilatations were done, the patient being treated by wet compresses. Suppuration occurred and the gland was incised 4 days later. Convalescence was uneventful.

CASE 16. J. G., aged 11 years, male. This patient was operated on for ruptured appendix with peritonitis on October 1, 1930. Culture of the peritoneal fluid showed the presence of bacillus coli. On entrance to the hospital he was very toxic and had a temperature of 102 degrees. Following the operation his temperature fluctuated between 102 and 104 degrees. He was irrational and was very difficult to handle, despite the use of morphine. His mouth was foul and was kept clean with great difficulty. At times he was incontinent of urine and fæces. On October 6, 1930, swelling of both parotids was noted and ice was applied immediately. The patient's temperature and general condition remained about the same. On the advice of a consultant, dilatation was omitted and treatment was confined to the use of hot, wet applications. Despite the parotid infection, the patient improved somewhat. On October 12, both glands ruptured into the auditory canal. The following day the glands were incised by small incisions just below the ear. A large amount of thick pus was evacuated and drainage instituted by rubber tubes. Staphylococcus albus was cultured from the pus. Convalescence was uneventful and the patient was discharged on November 25. The parotid wound was entirely closed in about 3 weeks. A lipiodol picture was taken prior to his discharge (Fig. 14).

GENERAL CONSIDERATIONS

In our first acute cases, we always instilled mercurochrome after the irrigation with normal saline. In our later cases, we omitted the mercurochrome and we feel that our results are as good. The mercurochrome seemed to cause some reaction which we feel is unnecessary in the acute cases. In the chronic cases, the use of mercurochrome, lipiodol, or some other mildly irritating substance is advisable. Dr. C. (Case 6) told us that following each instillation, the gland was

Fig. 11. Shows marked dilatation and pocketing of Stenson's duct. Gland not well visualized.

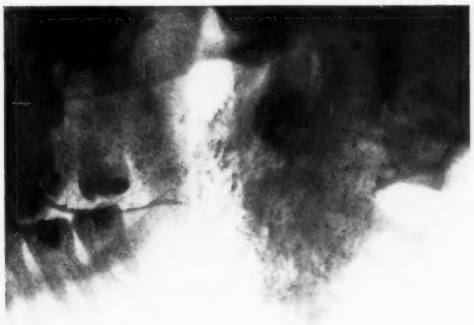

Fig. 12. Shot-like appearance of gland. Duct apparently normal.

tender for about 24 hours but that the interval of relief was greater than when dilatation and irrigation with saline alone was done.

Treatments were given not oftener than every 24 hours in the acute cases. Case 2 received treatments more often but the same result was obtained in our other cases treated less frequently and the patient was spared the discomfort of the procedure. In the chronic cases, treatment was given just prior to the recurrence of the symptoms (estimated) or when the symptoms recurred. Case 6 noticed that treatments at 6 week intervals kept him perfectly comfortable. The operator must use his own judgment in each case.

In reviewing the cases presented, we note several clinical facts:

First, all of the acute cases were ushered in by the classical syndrome of sudden acute swelling, associated with pain and rise of temperature.

Second, of the 16 cases presented, in 9 the orifice of Stenson's duct was pouting, red, and angry looking and thick mucopurulent material could be expressed on light massage of the gland and duct. Of these cases, 2 were comatose and the infection was merely an incident in a prelethal state (Cases 12 and 14). Treatment was therefore without effect. One case (Case 15) received but one irrigation, the treatment then being changed to the usual local applications. One (Case 16) received no treatment whatsoever. Thus, there were but 5 cases treated by the authors by dilatation and irrigation of the gland. These received daily treatments, and all subsided within 5 days. The case not treated by our technique throughout (Case 15) and the one (Case 16) receiving only local applications suppurated. One case (Case 2) received several dilatations the first day, but we deem that unnecessary.

Third, one case (Case 5) showed a normal duct orifice and secretion. This patient was aggravated by our treatment and required surgical intervention.

Fourth, of the remaining 6 cases, the 4 chronic cases all showed varying degrees of prominence of the orifice and some abnormality of the secretion. In these cases, our treatment carried out over a period of time, in 1 case (Case 6) over a year, resulted in a clinical cure. Thus in the chronic cases, showing a similar although less marked picture of prominence of the duct orifice and abnormality of the secretion, we may expect clinical cures when treated by our method.

In summary, all the cases reported in vital patients, whether acute or chronic, showing prominence of the orifice of Stenson's duct and abnormality of the secretion, were benefited by our treatment. The one acute case showing normal Stenson's duct orifice and secretion was made worse by our treatment.

At this point it seems wise to us to attempt to rationalize. Similarity between infections of the parotid and infections of the kidney have been mentioned in literature repeatedly. Since the advent of the cystoscope, the treatment of kidney infections has been fairly well standardized. Infections of the parotid are still treated in general according to Galen's idea of abscesses beside the ears. Infections of the kidney are roughly classed as infections of the parenchyma or kidney tissue proper, and infections of the

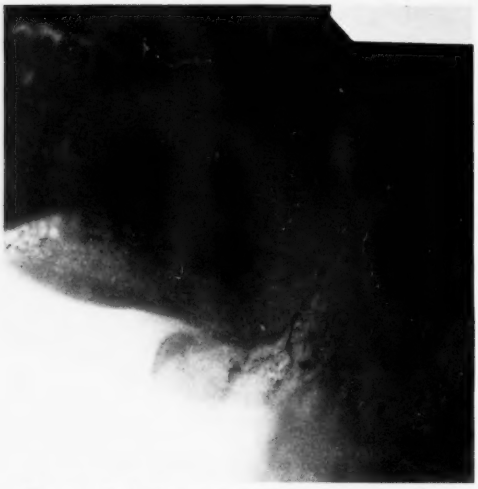

Fig. 13. Pocketing of ducts in gland. Stenson's duct apparently normal.

pelvis or drainage chamber. Parenchymatous infections either absorb or go on to abscess formation requiring surgical intervention. Infections of the pelvis, however, are treated by dilatation of the ureter and irrigation of the pelvis.

Surgical procedures are necessary only when the condition is too far advanced to respond to this type of treatment or when the parenchyma is involved as well. In infections of the parotid on the other hand, no attention has been paid to the normal drainage canal, Stenson's duct. Rankin and Palmer called attention to this type of drainage but consider it a matter of good fortune when this occurs, as it does, spontaneously at times. Keith[1] made use of this duct drainage and irrigation and his results correspond to ours. All other treatments consist of external applications of some sort.

Turning back to pyelitis in the kidney, there is no question but what the use of heat, alpine lights, possibly X-rays over the kidney region would relieve the condition at times; also the forcing of fluids and the use of urinary antiseptics. We know, of course, that very few cases of pyelitis are treated by drainage of the kidney pelvis. We feel certain, however, that were cystoscopic treatments painless and inexpensive, more patients would be treated by this method and, according to urologists with whom we have talked, fewer operations would have to be performed upon the kidney.

[1] J. Am. M. Ass., 1928, xc, 1270.

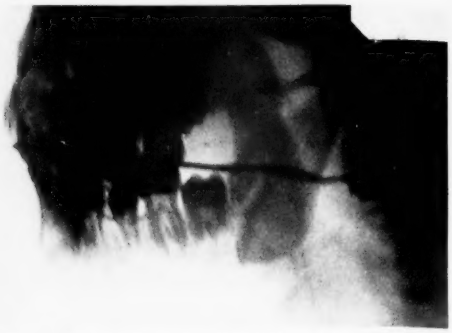

Fig. 14. Abnormal case. Dilatation and irregularity of Stenson's duct and ducts of gland. Patient treated by external applications only.

In pyelitis in the kidney, we demonstrate visual proof of our pathology by injecting a radio-opaque substance into the pelvis of the kidney. The pelvis may be distorted and the ureter at times shows narrowings in its caliber which if severe enough, are known as strictures. Although the structure of the parotid and the kidney are not similar, they are analogous. If our reasoning is correct, we should see abnormalities in the gland or pelvis as we call it, and in the caliber of Stenson's duct. The accompanying lipiodol-injection roentgenograms demonstrate this fact very well.

In the plates of normal glands (Figs. 1 to 5), we notice that the duct is smooth, the branchings clear cut, and the terminal ductules distinct. In our plates of infections of the pelvis of the parotid, we note a blurring, distortion, or actual obliteration of these outlines, a close resemblance to the difference between normal and abnormal kidney pyelograms. The amount of distortion in both conditions will depend upon the length and severity of the infection. In our chronic cases of which we were fortunate enough to have pictures made, we see the same condition. It must be clearly understood, however, that this explanation is not without its flaws. The parotid pelvis does not correspond exactly to a kidney pelvis and our plates therefore will not correspond exactly. In pelvic infections of the kidney, we often find distortions of the ureter. Our plates show this same condition in the parotid duct. We, however, only pretend to differentiate normal from abnormal conditions. Our data is too small for us to attempt to differentiate pathological conditions as we might in kidney infections.

Etiology. Our work does not embrace enough cases to warrant a positive statement as to

etiology. We personally agree with Jennings that either the ascending or the pyæmic type of infection may be present. From our work we feel positive, however, that those cases showing acute inflammation of the gland with a normal duct and normal secretion are pyæmic in origin. To our minds, it is like parenchymatous infection in the kidney. In the other acute cases, we felt that ascending infection plays the chief rôle, as here oral sepsis and lowered resistance are usually present. We are free to admit, however, that this does not hold true in all cases. Case 16, in which the cultures from the abscessed gland showed a different organism than that obtained from culture of the peritoneum would seem in favor of this view. But it is possible that secondary infection may have occurred.

CONCLUSION

In conclusion, we believe that our method of treatment of infections of the parotid gland, both in the acute and chronic cases, is a distinct advance upon the methods elaborated up to this time. It is physiological in that it drains the infection through a normal passageway. It differentiates between the type of infection which will respond to our method and that which will require surgical intervention. It requires no special instruments or unusual skill. It causes the patient little discomfort and gives a reasonable assurance of success.

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INTUSSUSCEPTION[1]

Surgical Treatment in Reducible Cases

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INTUSSUSCEPTION is said to be the chief cause of intestinal obstruction in children under the age of 5; from 50 to 75 per cent occur under 1 year of age.

It may be well to enumerate at least some of the etiological factors which have been advanced as causative agents of intussusception: (1) excessive mobility of cæcum and ascending colon; (2) narrowing of bowel at the site of intussusception; about 80 per cent occur at ileocæcal valve. According to John Fraser, of Edinburgh, a slight intussusception at the ileocæcal valve is a normal occurrence. (3) Irritability of the bowel as in enteritis with the increase in peristalsis. (4) Relaxed mesentery of the bowel, particularly the ileum. (5) Hypertrophied Peyers patch or intestinal polyp.

Generally acute intussusception is associated with considerable shock. In the early stages vomiting is usually present, with frequent mucus and blood stained stools. In about 70 per cent of cases a sausage shaped tumor can be palpated. In the beginning the abdomen may be scaphoid. But later distention is likely to supervene. Because the circulation in intussusception is impaired by the constriction of the bowel involved, eventually gangrene will result.

The prognosis, as in many other acute abdominal lesions, depends on early diagnosis and surgical treatment. Only a few years ago the mortality in these cases was quoted as high as 37 per cent even when the patient was operated upon within first 24 hours. When surgery was deferred as long as 3 or 4 days the mortality was as high as 65 or 70 per cent. Early recognition and prompt surgery are therefore very essential in the reduction of the mortality ratio. As most of these cases occur in early infancy, any operative procedure which prolongs the operation will naturally raise the mortality.

When the intussusception can be reduced, it must be accomplished as quickly and with as little injury to the bowel involved as possible. After the reduction is achieved, the surgical procedure must aim to prevent a recurrence of the intussusception. Some cases have recurred as many as four or five times.

The following operations have been suggested and performed to prevent recurrence:

1. Shortening of the mesentery of the ileum by a continuous Lembert suture so as to make a fold in it. In this operation injury to the circulation of the bowel is often unavoidable. Because of this factor and the longer time consumed in performing this operation, it is not advisable, especially when the infant is already very ill.

2. Suturing the omentum to the ileum at the ileocæcal junction is also recommended. This operative procedure has its merits and may be quite effective in preventing recurrence. Its chief drawback is that it might cause an uncomfortable tugging at the transverse colon and stomach.

Fig. 1.

Fig. 2.

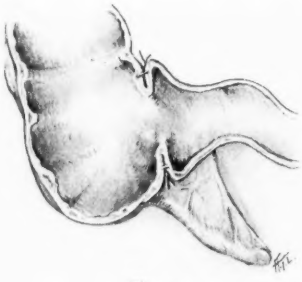

Fig. 3.

Fig. 1. Intussusception before reduction.
Fig. 2. Intussusception reduced. Method of suturing ileum to cæcum.
Fig. 3. Cross section showing sutures in place and outfolding of ileum over the cæcum.

[1]Presented before The Pacific Coast Surgical Association, Seattle, Washington, February, 1931.

However, we often find the omentum attached to an abdominal scar in the lower abdomen without causing any noticeable symptoms. I would give preference to the operation that will prevent recurrence without deliberately fixing the omentum to the ileum.

3. Suturing the terminal ileum to the parietal peritoneum with two or three sutures to prevent recurrence. This may be an effective method of preventing recurrence, but it seems that the fixation of the ileum may interfere with normal peristalsis and give a considerable amount of trouble to the patient.

4. Suturing the terminal ileum to the cæcum and ascending colon for a distance of 5 or 6 inches has also been tried. This operation, too, will prevent recurrent invaginations of the bowel, but it may also interfere with normal peristalsis.

5. Some surgeons suture the cæcum and ascending colon to the parietal peritoneum to prevent the excessive mobility of the cæcum and ascending colon which is considered one of the chief factors in causing intussusception.

Another method in the surgical treatment of intussusception occurred to me while operating recently on an infant 5 months old. I believe it has some additional merits. Besides preventing recurrence it does not alter the anatomy radically. Many of the operations that have been done for intussusception are prone to cause disagreeable symptoms necessitating a second operation in order to undo the mischief done by the first operation.

This operation which may be applicable to other areas of the intestines, is suggested particularly for intussusception at the ileocæcal region which is the site of occurrence in the majority of cases. Since there is present normally a slight degree of intussusception at the ileocæcal valve—an infolding of the ileum into the cæcum, the operation suggested is the suturing of the ileum to the cæcum with three or four silk sutures in such a way as to cause an outfolding of the ileum on the cæcum (see illustrations).

I am of the opinion that this simple procedure accomplishes all that the more elaborate and lengthy operations do and it can be done in 2 or 3 minutes. This time element is an important factor in lessening surgical shock which too often is the cause of unfortunate results. In addition to this operation, if the cæcum and ascending colon are abnormally mobile, they should be fixed to the parietal peritoneum by three or four sutures as suggested by Sir Berkeley Moynihan.

EPILEPSY

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FOR many years epilepsy has been considered a degenerative disease of the brain. The degenerative changes have been supposed to cause atrophy of the brain, resulting in cortical irritation which, in turn, causes the convulsive seizure. Pathologists, studying the subject, have confined themselves almost entirely to the brain; physiologists have been unable to explain the peculiar phenomenon of the convulsive seizure. Treatment has been directed largely toward alleviating the spells by diminishing the irritability of the cortex of the brain with drugs. Thus the patients have been allowed to go on indefinitely with little or no benefit, and a gradual mental deterioration, ending in either suicide, death in some institution, accidental death due to the seizure, or death in status epilepticus, has been the picture of epilepsy through the ages.

Today, as the result of clinical observation and research in the physiology and pathology of the brain, particularly as applied to the cerebrospinal fluid, water metabolism, and the anomalous dural venous sinuses, an entirely new and different picture appears. Just as it was formerly believed that the glial formation caused the gradual contraction of the cortex, resulting in convulsive seizures, so it was also believed that the sole function of the cerebrospinal fluid was to act as a buffer or cushion for the protection of the brain and to carry off the by-products of neurometabolism. The fallacy of this conception of the cerebrospinal fluid, shown by the work of Gamble, completely changes this idea. Moreover, it demands a change in the theory regarding the atrophied brain. If the atrophied brain is the result of accumulation of cerebrospinal fluid acting as a hydraulic press—as has been shown conclusively by Fay and Winkelman—the ischæmic atrophy is caused by pressure and not by shrinkage of the glial tissue. In other words, it seems that the water cast (Fay), which surrounds the outer surfaces of the cerebral hemispheres and which extends into the cisterns and ventricles as well, produces the pressure atrophy by hydraulic action.

Under all circumstances the convulsive seizure requires both an intact motor pathway[1] and, also, a gradual irritation over a large surface of the cortex, particularly over the motor areas. With this gradually increasing irritation to the cortex, it needs only a sensory stimulus to set off the massive reflex action which constitutes a convulsive seizure. Just as the simple reflex caused by tapping the knee with the finger is a normal response, so is the massive reflex of a convulsive seizure a normal response. The only difference between the flexion and extension motions of the runner as he crosses the tape in a hundred meter race, and the successive contractions and extensions in a convulsive seizure, is that one is purposeful and the other is not (Fay).

[1]Ito, 1912; Foerster, 1926; Dandy and Elman, 1925; Speransky, 1926; Elsberg and Pike, 1926; Cobb, 1928; Pollock and Davis, 1927.

WATER METABOLISM

The research of Gamble, Ross, and Tisdall on water metabolism has led to the solution of many problems in the study of epilepsy and has placed clinical observations upon a scientific basis. Gamble has been able to show that water metabolism plays a most important part in diseases in which œdema or dehydration occurs. Briefly, he has demonstrated that of all the water in the body, approximately four-fifths is intracellular in character, bound in the cells by a fixed potassium base. The other fifth is divided almost equally between the vascular and the interstitial water, both being bound by a fixed sodium base. The vascular water is utilized in the ordinary metabolic processes and is replenished from the interstitial reservoir (Fig. 1). When more water is demanded for metabolic purposes, it is readily supplied from this same reservoir, the largest accumulation of interstitial water being situated in the ventricles and subarachnoid spaces of the cranial cavity. Evidently this reservoir is placed in the safest part of the body, the bony skull, for the same reason that the brain itself is placed there, i.e. both are absolutely vital to life. Furthermore, this water must be accessible for instant use by the blood stream, and its situation in the cranial cavity fulfills this requirement.

In addition to the work of the above men, Drabkin and Shilkret, 1927, and Ravdin, 1929, have further shown that it requires a smaller dose of insulin to produce a convulsion in a hydrated animal than in one dehydrated. Another recent contribution by Lawrence and Rowe shows that the anterior pituitary lobe may be considered as the motor activator of the ovaries, as well as a factor in water storage. This offers an explanation of the storage of interstitial water for the menstrual

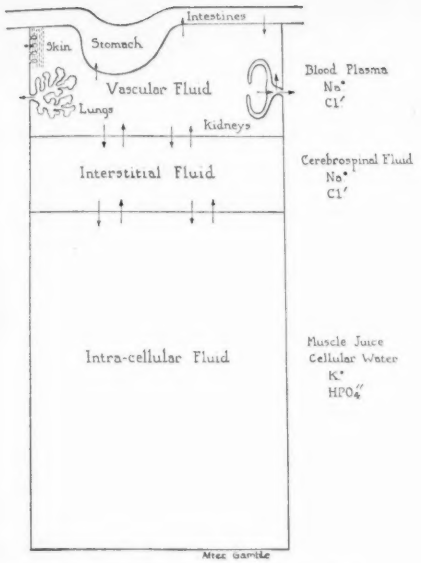

Fig. 1. Distribution of fluid in the body.

cycle. Berens (working in my laboratory) will soon publish clinical studies on this phase of epilepsy.

These workers have given us a new conception of the importance of water metabolism and the functions of the ventricles of the brain. Gamble (1929) has shown exactly what occurs during the convulsive seizure. The rising tide of cerebrospinal fluid having gradually reached its climax, the basic sodium salt and water enter the venous channels and the circulatory system, and are eliminated largely through kidney excretions, perspiration, and respiration. Afterward, the cerebrospinal fluid pressure in the interstitial spaces returns to normal, and the cycle is repeated. Gamble, therefore, substantiates Fay's idea of a water cast causing a gradually increasing irritation over the cortex of the cerebral hemispheres. It is not possible to say at this time just what touches off the mechanism. S. Kinnear Wilson believes that it is neuronic in origin; Spiller and Fay share this belief. Lennox and Cobb (1928) add decreased oxygen tension as another factor in producing the convulsion. It may be true that any sensory stimulus could initiate the convulsive seizure. Furthermore, added pressure itself may be the chief factor.[1]

[1]Lind described thickening of the arachnoid and traction by the trabeculæ producing a pulsating pull on the cortex as a possible source of subminimal stimuli amounting to irritation.

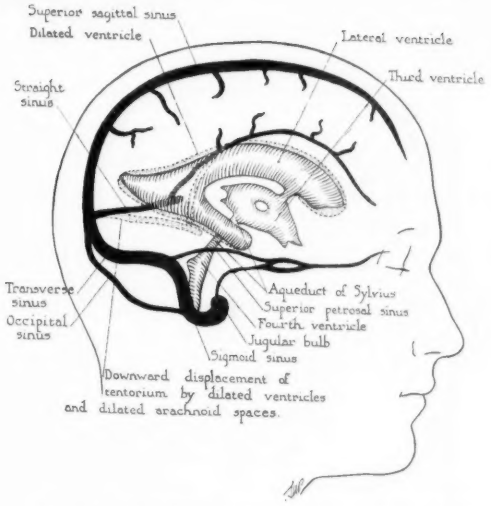

Fig. 2. The cerebrospinal fluid forms in the ventricles as shown in the outline in the center, passes from the lateral ventricles to the third ventricle, through the aqueduct of Sylvius to the fourth ventricle and outward to accumulate in the basilar cisterns and over the cortex of the brain. The dark lines show the venous dural sinuses. The cerebrospinal fluid enters the sinuses above and courses backward then downward and lateralward on either side, to leave the skull at the jugular foramen. The shaded area shows the displacement of the straight sinus, which corresponds to the plane of the tentorium, whenever there is a dilated ventricle or increase in the volume of brain substance, tumor mass, or accumulation of fluid in the subarachnoid spaces over the cortex.

To summarize, it is possible to say that water metabolism has an important rôle in the initiation of periodic convulsive seizures; that the accumulation of fluid in the subarachnoid spaces, cisterns, and ventricles of the brain brings about a constantly increasing irritation to the cortex of the cerebral hemispheres, particularly over the motor areas; and that this increasing pressure finally creates such a degree of irritation that the simplest sensory stimulation, even though psychic, may set off the actual convulsive seizure. Following the convulsion there is an elimination of fluid, largely through the kidneys but also through the skin and respiratory pathways. Modern research has established these factors as essentially the set-up for the convulsive seizure. Fay, in discussing the part water plays in the convulsive state, says:

"Viewed in the light of our experience with the acute and chronic convulsive state we have been forced to accept the fluid factor as a most important *predisposing* agent *influencing other mechanisms responsible for the seizure itself.*"

THE CIRCULATION OF THE CEREBROSPINAL FLUID

After leaving the cisterns and subarachnoid spaces the cerebrospinal fluid normally filters easily through the Pacchionian bodies into the superior longitudinal sinus, and thence, by way of the transverse and sigmoid sinuses, jugular bulbs and jugular veins, it reaches the heart (Fig. 2). If, however, the Pacchionian bodies are faulty in development or are diseased (Fay and Winkelman), if the venous sinus is faulty in development,[1] if the jugular foramina are too small, or if there is sufficient pressure from any cause to obstruct the easy flow in the jugular veins, the cerebrospinal fluid becomes blocked, first in the subarachnoid spaces and finally in the ventricles. Ordinarily, large quantities of cerebrospinal fluid are stored in the reservoirs of the ventricles, cisterns, and subarachnoid spaces ready for rapid withdrawal at any time, but in conditions of faulty development, or in those resulting from trauma or inflammation, this mechanism is disturbed.

VENOUS DURAL SINUSES—
NORMAL AND ANOMALOUS DEVELOPMENT

Channel selection and rudimentary sinuses. Streeter has not only shown that the normal sinus development is symmetrical, but that in some cases the right transverse sinus drains the superior longitudinal, while the left transverse drains the straight sinus. This latter fact in itself is not important except that it shows that a variation from the normal symmetrical development is not uncommon. When the developmental process is complete, even though the above variation occurs, there is usually a confluence of the channels at the torcular Herophili and the blood may take either course as occasion demands.

Streeter uses the term "channel selection" in describing the peculiar selective process during the embryological development of the sinuses. By that he means the tendency for one vessel to become dominant among a newly formed group, while the others, by coalescence or even by actual obliteration, are lost in the migratory process occurring in the growth of the brain, dura, and sinuses (Fig. 3). Undoubtedly, it is this selective process, so well described by Streeter, that accounts for the many variations in the caliber, position, and even the number of sinus channels found in epileptics. That these anomalies are present in a very considerable number (Table IV) suggests their agency in the disturbed venous drainage in epileptic cases. The most common variations from the normal symmetrical system are the large right and the smaller or rudimentary left transverse sinus (Fig. 4). Of less frequent occurrence are the dilated sigmoid sinuses which I believe are always associated with small jugular foramina (Fig. 5). In certain cases this dilatation is well established at the time of birth. In two autopsies where death occurred within 48 hours of birth, there was a splitting apart of the dural walls of these sinuses (Fig. 6), the jugular foramina being too small to permit sufficient drainage. Both infants died in status epilepticus. X-ray pictures clearly show these sigmoid sinuses if the latter are largely dilated and, as Pendergrass has demonstrated, even the size of the jugular foramina can be determined. Other anomalies have been described in a former paper (Swift, 1929).

In a series of operations upon epileptics, I have found anomalous venous dural sinus development in 20 per cent of the cases. This would clearly establish a hereditary or a developmental factor as the cause of certain cases of essential epilepsies. This is in accord with the findings of S. A. Kinnear Wilson that only 16 per cent of epilepsies could be traced to a true hereditary background. I believe that these two groups of cases are identical in etiology, due to a developmental defect rather than hereditary. This faulty development predisposes to mechanical obstruction, due to the peculiar circulation of the cerebrospinal fluid.

Mechanical obstruction of the venous sinuses. Fluids circulating in a closed cavity do not increase in volume but only in velocity under variations in pressure conditions. In other words, the amount of blood in the venous channels does not vary in volume under normal conditions. Thus an increase of intracranial pressure is compensated for by an increase in the velocity of flow within the venous channels (Monro-Kellie doctrine). Three elements are factors to be considered in any change from the normal: the brain, the blood, and the cerebrospinal fluid. If there is, for instance, an increase in one, the brain substance, the initial effect will be an increase in the velocity of flow of the blood, while a further increase in brain content will displace the third element, the cerebrospinal fluid. Since the cerebrospinal fluid is displaced by way of the venous channels, it follows that any obstruction to the venous outflow will retard the displacement of this cerebrospinal fluid. This will in turn act as a further cause of increased pressure. Since the reservoirs are largely above the tentorium, this increased pressure causes a downward displacement of the tentorium (Fig. 2, dotted lines), thus compressing the walls of the transverse sinuses and decreasing the caliber of the venous channels (Fig. 7). As the caliber decreases and the amount of cerebrospinal fluid increases, a higher

[1]Swift, 1930.

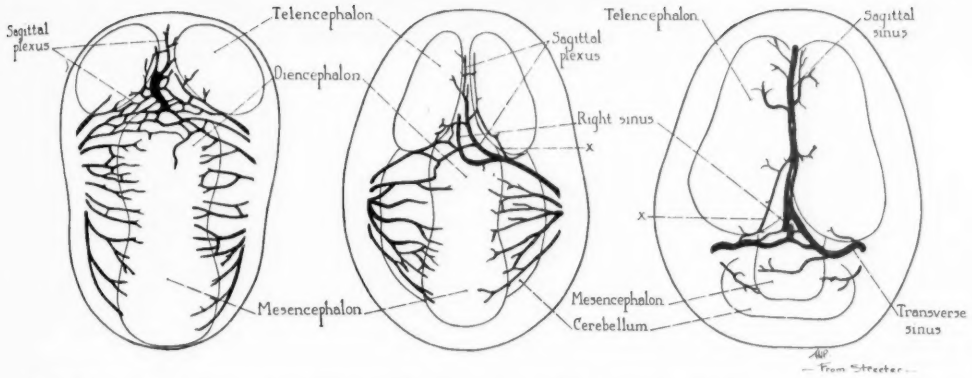

Fig. 3. Diagram after Streeter showing how the developing brain causes a coalescence in the midline to form the superior longitudinal sinus and how it causes the migration and coalescence of the transverse primary head veins to form, by channel selection, the right and left transverse sinuses.

intracranial pressure results. Fay, in a personal communication, states: "Thus, if there is a ventricular block, the pressure should be sufficient not only to force the cerebellum down through the foramen magnum, but against a collapsible sinus. Pressure must be equal on both sides of the tentorium and the only point of release is the venus sinus." This is illustrated in Figure 7. There must of necessity be some displacement downward and backward of the dural wall lateralward to the median line, while none occurs at the median line where the straight sinus is found. Mechanically the set-up is perfect for a convulsive seizure. Then as the head is thrown backward and the jugulars are placed in their optimum position for venous drainage, the venous blood is carried away, the cerebrospinal fluid pressure is relieved, and the set-up for a convulsive seizure is dissipated. Extensive study of defective venous drainage led to the development of the operation which will be described later. I find that by mobilization of the transverse sinus it becomes impossible to compress the sinus when the tentorium is displaced downward.

As stated, Fay and also Gamble have shown that a gradually increasing amount of cerebrospinal fluid is essential for the convulsive set-up. A logical explanation for the retardation of the cerebrospinal fluid outflow by way of the venous channels is found in the faulty development of the venous sinuses. This retardation may occur as a result of blocking at any of the following points: the Pacchionian filter, the longitudinal sinus, the transverse sinus, the sigmoid sinus, the jugular bulbs, the jugular foramina, or the jugular veins (Fig. 8). Unless there is such a partial obstruction, an increase in the cerebrospinal fluid may be easily adjusted by free drainage through these venous channels. Because the factors which govern the transverse sinus are not found in any other sinus—namely, variations in caliber due to increased pressure—it follows, therefore, that the transverse sinuses become the important factors in what may be termed the threshold in epilepsy. Increase in the caliber of these sinuses above normal tends to decrease the possibility of a convulsive seizure; on the other hand, decrease in the caliber tends to increase the possibility of seizure. The greater the obstruction of the venous outflow, the less the load required to produce a convulsion. Senile epilepsy is an excellent illustration of a mild degree of decreased venous drainage, while that of young patients in which the jugular foramina are small but patent, with dilated sigmoid sinuses above, illustrates the severe type. In the latter the brain is markedly atrophic, and nothing entirely controls the seizures. Even constant spinal drainage gives only temporary relief; almost all die before reaching the age of 7 years. Midway between the senile and the infantile types is that of women whose convulsions, having begun with the menstrual cycle, diminish or cease temporarily during pregnancy and stop at the menopause.

A factor of importance should be mentioned here, namely, the effect of the position of the body upon venous drainage. Lewis has demonstrated that the venous drainage reacts promptly to any changed position of the head or body. He has been able to show, by means of an intricate mechanism for measuring the cerebrospinal fluid pressure, that variations in pressure cause changes in

TABLE I.—PATIENTS OPERATED UPON FROM OCTOBER 3, 1925 TO NOVEMBER 23, 1927—33 CASES

Case	Age	Age at onset: Petit mal	Age at onset: Grand mal	Mental deterioration (—)	Encephalography: Date of examination	Encephalography: Pressure cerebrospinal fluid	Encephalography: Ventricular dilatation	Encephalography: Cortical air	Results of original operation: Date of operation	Degrees of improvement (+), no improvement (—), in attacks and mentality: Petit mal	Degrees of improvement (+), no improvement (—), in attacks and mentality: Grand mal	Degrees of improvement (+), no improvement (—), in attacks and mentality: Mentality	Cured	Comments; deaths; time since operation
	yrs.	yrs.	yrs.			mm.								
1	2½		2½		10–1–25	35	+++		10–3–25				+	
2	1½		1½		10–28–25	20	+		10–21–25					
3	4		6 mos.	— —	8–25–24	26		++	11–17–25		+			
4	13	Birth		— — —	1–3–26	30	+++		1–27–26		++	+		Recent report: no spells past year; in school.
5	21		15 mos.	—	4–1–26		+++		4–20–26		++			
6	5		3½	— —	4–22–26	26	+		4–24–26		+			Recent report: no spells past 2 years.
7	4	3½	3¼	— —	4–19–26	22	+++		4–26–26		—	—		
8	1½	4 mos.	4 mos.	—	1–9–28		++		5–1–26		—			
9	10	8½	8½	—	5–14–26	42	R +++		5–27–26					Recent report: no improvement.
10	1½	6 mos.	8 mos.		7–13–26	28	+++		7–19–26	—	+			
11	23	20	20		7–22–26	28	++++		7–31–26				+	Recent report: no spells past 2 years.
12		10		— — — —	8–19–26	40	R ++		8–21–26			+++		Recent report: marked improvement mentally.
13	3½	2½		— — — —	8–26–26	60	++		9–9–26					
14	22	Birth	2 mos.		9–3–26	20		++++	9–14–26 7–10–28		+			
15	23	19	19		9–25–26	20	++++		9–28–26		+			Died 12–9–27; accidental death. (9 mos.)
16	2	3 mos.		— — —	11–20–26	46	R ++	+++	11–27–26 7–7–28 1–3–28	+++				Marked improvement in petit mal.
17	22	3	3		9–23–26	18		++++	11–30–26	+	+			
18		Birth			12–13–26	14	+++		12–16–26					
19	14	6½mos.	6½mos.	— — — —	1–6–27	30	++		1–8–27					
20	2		Birth	— — — —	1–30–27	24	+		2–10–27 8–6–28	+	+			
21	10½	4 mos.		— — — —	2–8–27	28			3–5–27			+		
22	4		2	— —	11–17–27	20	++		11–23–27	—	+			
23	21		21		3–21–27 1–2–29	30 40	++++ L ++	R ++	4–12–27		—	++		
24	16		7		2–17–27	30			4–16–27		—	++		
25	5	18 mos.	18 mos.		4–19–27	40	L +		4–26–27	+++	+++	+++		
26	5		18 mos.		4–19–27	28	++++		11–2–27 4–25–27		+			
27	10½mos.	4½mos.	4½mos.		4–30–27	24	+++		5–27–27 8–4–28	+	+	+		
28	8		5½mos.		4–23–27	40	L +	++	6–22–27	+++	+++	+++		
29	16		18 mos.		6–21–27	30	++	+	6–23–27		+	+		
30	13		13		7–7–27	30	+	++	8–1–27					Died on 2nd postoperative day; hæmorrhage.
31	14		4						8–1–27					
32	1		3 mos.		8–17–27	20	+++		8–20–27					Died 11–30–27; pneumonia (3 months.)
33	1¾		17 mos.			38		+++	9–2–27				+	
Average 10 yrs.					Average 28 mm.				Totals	6 + 2 —	15 + 4 —	9 + 1 —	3 +	3 deaths.

the venous return. These changes are caused by the position of the patient, flexion of the head, or the slightest pressure on the jugulars (Lewis).

Summary. Anomalous development and mechanical obstruction of the venous sinuses show that periodic convulsive seizures occur in the human, only when there is some interference with the outflow of the cerebrospinal fluid through the venous dural sinuses and thence to the heart. This obstruction may occur anywhere from the Pacchionian bodies to the heart itself. Under normal developmental conditions the dural venous sinuses can and do carry a variable amount of cerebrospinal fluid depending upon the metabolic processes of the body, the variations in water storage, as in the menstrual cycle, and the pathological changes associated with water metabolism incident to hydræmia in acute alcoholism, acute infections, and poisons. Anything that prevents this easy variability in circulation of cerebrospinal fluid, such as inflammatory conditions of the arachnoid, or diseased conditions of the Pacchionian bodies, may cause the set-up for a convulsive seizure when normally developed sinuses are present. In approximately 18 to 20 per cent of cases, however, anomalous or faulty development of the transverse dural sinuses, or the occipital bone and its jugular foramina, prevents this normal venous drainage. In these cases certain physiological conditions, such as an accumulation of cerebrospinal fluid, "may cause an anoxæmia of the frontal lobes which so lowers the threshold as to permit a sensory stimulus to explode the motor areas in the same way that loss of cortical inhibition and control of the lower motor neuron permits a more prompt and active response to a small stimulus in the tendon reflex" (Fay).

Thus it would appear that the hereditary influence in epilepsy, as pointed out by S. A. Kinnear Wilson, shows itself in this series of developmental defects of the transverse sinuses and occipital bone (Table V). Furthermore, while many of these cases may not have defects sufficient to cause more than a transient seizure during pregnancy or acute infection, or even to become manifest until arteriosclerotic changes of senility develop, they are, nevertheless, potential epileptics. That venous drainage is the essential factor, not only in epilepsy but also in the development of the sudden intracranial disturbances which occur in brain tumors, choked discs, acute dementias, et cetera, cannot now be questioned.

FIXATION OF THE TRANSVERSE SINUS

A knowledge of the attachments of the sinuses is important in understanding the mechanical factors involved in the narrowing of the lumen of the transverse sinus (Fig. 9) At the point known as the torcular Herophili, a large emissary vein leaving the sinus system, attaches the sinus firmly to the skull. There may be several small emissary veins, and there is invariably a deep groove in the skull at this point. As the sinuses extend laterally to their junction with the sigmoid, the two dural layers, which split to form the transverse sinus, again become confluent and extend forward along the margin of the petron. The small, triangle-shaped superior petrosal sinus is situated between the two layers of the dura and joins the transverse sinus at its junction with the sigmoid, at a point which is always fixed (Fig. 9). This makes an angular formation of the tentorium at the superior posterior portion of the mastoid, and gives a fixed point on either end of the transverse sinus. Pressure from above downward, or displacement of the tentorium from above downward, collapses the lateral walls of the sinus and thus causes a narrowing at the angle where the sigmoid joins the transverse sinus. This is easily demonstrated on a cadaver. The slightest pressure from above downward will force the volume of fluid within the sinus upward into the superior longitudinal sinus with equal pressure. A variation in the sinus caliber will change the intravenous pressure to one equal or higher than the subarachnoid pressure. Weed, in experiments upon animals for the purpose of determining the effect of changed position of the body upon intracranial and intraspinal subarachnoid pressure, found the intravenous (dural) pressure curve to correspond with the subarachnoid curve—though always slightly below in actual measurement.

This fixation of the transverse sinus at the torcular Herophili and at the mastoid margin undoubtedly accounts for the increased pressure caused by tumors, either just above the tentorium in the occipital region or below in the cerebellar region. In the latter instance, however, the displacement is upward, also much less, due to the dense midline dural band which extends from the torcular downward to the margin of the foramen magnum. This band frequently carries a large venous sinus, the occipital sinus. Nature has provided, furthermore, a series of rib-like folds along the upper and lower surfaces of the transverse sinus, at right angles to the sinus wall, much like the knees of a ship on the under surface of a deck. In some cases these folds are highly developed; in others, there is practically no development. The falx cerebri coming down from above joins the tentorium and also assists in strengthening the vessel walls. In epileptics, however, the folds of

TABLE II.—GROUP 2—SIXTY-FIVE CASES—MORE EXTENSIVE OPERATIONS

Case	Age	Age at onset: Petit mal	Age at onset: Grand mal	Mental deterioration (−)	Encephalography: Date of examination	Encephalography: Pressure cerebrospinal fluid	Encephalography: Ventricular dilatation	Encephalography: Cortical air	Results of improved operation: Date of operation	Degrees of improvement (+), no improvement (−), in attacks and mentality: Petit mal	Grand mal	Mentality	Cured	Deaths; time since operation
	yrs.	yrs.	yrs.			mm.								
1	29		8	– – – –					10–26–27		+++	+++		
2	6			– – – –	9–21–27				10–26–27		++			Apr. 1930; pneumonia (2 yrs.)
3	2		4		7– 3–24				10–28–27				+	
4	6		Birth	– – – –	11–12–26	28	++		10–29–27	−	−			Feb. 3, 1930; cause unknown (2 yrs.).
5	23		7	– –	10–24–27	28			10–31–27		++	++		
6	4½	Birth			10–28–27	34		+	11– 1–27	+		++		
7	13½		18 mos.		9– 9–27	38			11–11–27				+	
8	13		11						11–21–27		+++	++		
9	23		14		11–18–27	28	+	++	11–29–27				+	
10	24		17	– – –	11–20–27	28		++	11–30–27		−			
11	10	9½	9¾		12– 7–27	34		+	12–13–27		+			
12	7	6		–	12–17–27	24	++		12–20–27					
13	6		4	–	12– 7–27	30			1– 6–28		++	++		
14	13		11		1– 7–28	32			7– 2–28		−	−		
15	35		20		1– 7–28	24		+	1–16–28				+	
16	9	8			10– 7–27	28			2– 2–28					
17	27		17	–	7– 5–27	30	+	++	2– 2–28		+++	+++		
18	10	2½			2–14–28	48	+		2–29–28	+				
19	22	15	17		1–10–29	22		+	2–29–28	+	++	++		
20	7		3 mos.		3– 3–28	36	+++	+	3– 6–28					
21	47		23		6– 1–27	30			3– 8–28					
22	21		4		3– 9–28	32	+	+	3–13–28					
23	6		6 mos.		3–17–28	30	+		3–20–28		+	+		
24	6	1			3–23–28	24		+	3–26–28			++		
25	21		14		3–17–28				3–27–28					Nov. 11, 1928; suicide (8 mos.).
26	4½	6 mos.			6– 7–28	30			4–10–28	+++		+++		
27	7			– – – –	8– 2–28	32		+	9– 6–28			−		
28	1⅗	1			4–12–28	20	+	+	4–16–28					Apr. 19, 1930; status epilepticus.
29	3	20 mos.		– – – –	4–20–28	20			4–23–28					Died on 1st postoperative day; acute dilatation of stomach.
30	13		10		4–26–28	22		+	5– 2–28					
31	31		14		4–30–28	22	+	+	5– 2–28					
32	25		23		5– 8–28	26	+		5–11–28					Apr. 26, 1930; suicide (2 yrs.).
33	16		8		5–17–28	36	+		5–21–28		−	+		
34		7½	5 da.		5–17–28	32		+	5–22–28		−	−		
Average 15 yrs.					Average 29 mm.				Totals	6+ 3−	25+ 10−	28+ 4−	7+	10 deaths

TABLE II.—GROUP 2—SIXTY-FIVE CASES—MORE EXTENSIVE OPERATIONS—*Continued*

Case	Age	Age at onset		Mental deterioration (−)	Encephalography				Results of improved operation				Cured	Deaths; time since operation
										Degrees of improvement (+); no improvement (−), in attacks and mentality				
		Petit mal	Grand mal		Date of examination	Pressure cerebrospinal fluid	Ventricular dilatation	Cortical air	Date of operation	Petit mal	Grand mal	Mentality		
	yrs.	yrs.	yrs.			mm.								
35	11		1	−	5-25-28	42			7- 7-28		+			
36	7	6 mos.	3		7-11-28	40		+	7-16-28					
37	15		6		7-26-28	50			7-28-28		−	+		
38	24		14		8-16-28	30		+	8-18-28					Apr. 29, 1930; suicide (2 yrs.).
39	21		16	− −	8-16-28	25		+	8-20-28	−	++	+		
40	9	6½			9- 6-28	40		+	9-10-28		+	+		
41	3	5 mos.	1½		3-21-28				9-13-28	+	+	+		
42	26		16		4-17-28	28		+	9-18-28	+	−			
43	21		4		8- 1-28	32		+	9-24-28		+++	+++		
44	15		11		9-19-28	30			9-25-28				+	
45	10		6	−	9-20-28	30			9-26-28	−	++	++		
46	3		1		11-13-28	20			11-16-28					July 1930; drowned (1½ yrs.).
47	8	6 mos.	1		12-28-28	30	+	+	12-31-28	+		+++		
48	22		17		2-21-28	24		+	1- 8-29					
49	27		22		1- 2-29			+	1-11-29		+			
50	5		3½	− −	5- 1-29	20			7-17-29		++	++		
51	14		12	−	2- 6-29	20			2-19-29		−			
52	3		9 mos.		11-22-28	16			3-11-29		+	+		
53	25		19		3-11-29	30		+	3-18-29					Aug. 1929; cause unknown (8 mos.).
54	17				4-20-29	30			5- 2-29		+++	+++		Died on operative day; status epilepticus and serous meningitis.
55	10		3		8-23-28	34	+		5- 1-29					
56	28		14	− −	4-15-29	15		+	6-17-29		++	+		
57	9½		9	− − −	6-18-29	20			6-22-29		+	++		
58	22		14	− − − −	7-16-29	24		+	9-13-29				+	
59	18		3	− −	7-27-29	20			7-29-29		++	+		
60			18	−	8- -29		+	+	9-12-29		−	+		
61	13		6	− −	7-24-29				10- 2-29		+	+		
62	8		6		7-22-29	30	+	++	10-14-29			+		
63	23	5	8	− − − −	4- 4-29	30	+		11-18-29		+	+		
64	20		12		11-26-29	20		+	12-11-29		−	−		
65	1 7/12	7 mos.	1		5- 1-29	22	++	++	7-17-29				+	
Average 15 yrs.					Average 29 mm.				Totals	6+ 3−	25+ 10−	28+ 4−	7+	10 deaths

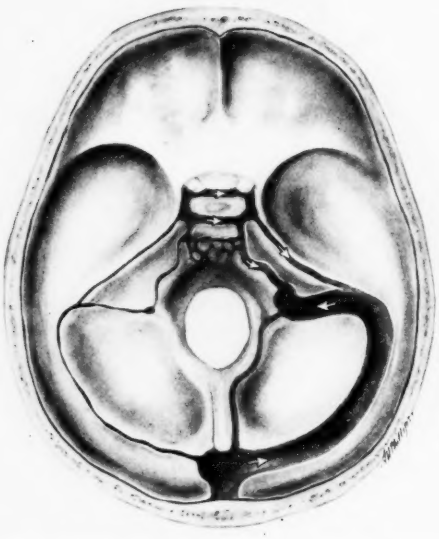

Fig. 4. Asymmetrical development of the basilar sinuses. Left transverse, rudimentary. Left superior and inferior petrosals do not function.

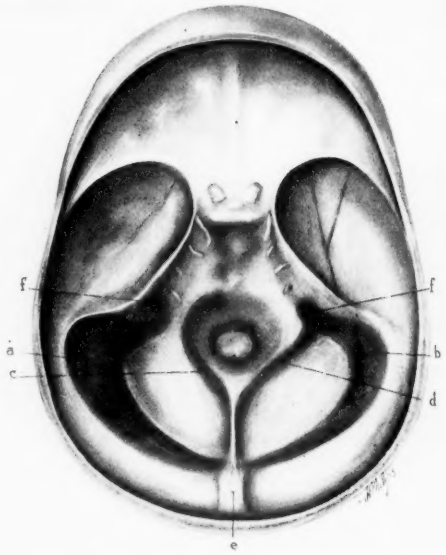

Fig. 5. This patient died at the age of 5 years in status epilepticus. The autopsy showed: *a*, dilated left transverse sinus; *b*, dilated right transverse sinus; *c*, left occipital sinus draining into an anomalous vertebral vein; *d*, right occipital draining into the right jugular bulb; *e*, dense fibrous dural band from the torcular to the posterior margin of the foramen magnum; *f*, small jugular foramina.

the dura are conspicuously absent, and the anomalous development of the sinuses prevents the symmetrical protective forces of the falx cerebri. Frequently, the superior longitudinal sinus balloons out well to the right of the falx at its attachment with the tentorium, and in such a case any pressure from above has a marked tendency to collapse the sinus (Figs. 10 and 11). These conditions have been described elsewhere (Swift). In these abnormal conditions collapse of the sinuses must be prevented if we hope to obtain free cerebrospinal fluid circulation. This is made possible by the mobilization of the transverse sinuses.

MOBILIZATION OF THE TRANSVERSE SINUSES

Souttar reports that, in operating upon the cerebellum for tumors, he found that mobilization of the transverse sinus permits the operation upon the cerebellum with ease. I have also found this to be true, and Figure 12 shows exactly what occurs. In this instance a large acoustic nerve tumor had caused an intense intracranial pressure. When the plate of bone overlying the torcular area had been removed, the distended sinuses could easily be seen. After puncture of the ventricle, the sinuses flattened; and at the second operation 3 days later, they were still flat, the pressure having been relieved. It is this mobilization of the transverse sinus over an extensive area, in fact out to the margin of the mastoid, that prevents a collapse of the sinus walls in the type of epilepsy just described.

Mobilization of the transverse sinuses then becomes of the utmost importance. Obviously, this is of no benefit if there is an obstruction at the jugular foramina or lower in the venous drainage system. Only in those cases which have the anomalous development or partial occlusion of sinuses is it essential for the transverse sinuses to be mobilized.

INDICATIONS FOR OPERATION

Application of the theory just advanced demands the observation of a certain regimen. Every case of epilepsy should have an encephalogram. The technique as described by Pendergrass should be followed; if the X-ray pictures reveal bilateral enlargement of both transverse sinuses or a marked enlargement of the right without any particular enlargement of the sigmoid as it enters the jugular bulb, the condition is one that can be markedly improved by mobilization of the transverse sinus. Such cases always show atrophy of the frontal lobes of the brain.

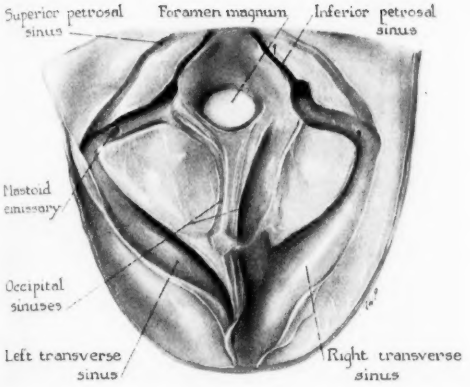

Fig. 6. The tentorium has been almost completely removed, and the sinuses open longitudinally. The pressure in the right transverse sinus would appear to have split the layers of the dura so that an enormously distended sinus wall develops. To a less degree, this is also true of the right occipital sinus. On the left side dilatation shows above but not so markedly below.

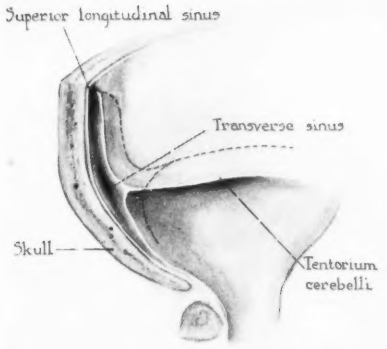

Fig. 7. Schematic section to the right of the torcular showing compression of the transverse sinus with displacement downward of the tentorium cerebelli. Dotted lines show the normal position of the tentorium and the interior wall of the sinuses.

CONTRA-INDICATIONS

Mobilization has not proved successful in cases of marked mental deterioration, nor has it proved beneficial to those patients in whom there is marked cerebral atrophy, particularly over the occipital as well as the frontal lobes. In my experience these cases are usually associated with very small jugular foramina.

Other contra-indications include traumatic epilepsy with localized accumulations of cerebrospinal fluid. An operation over the affected area is indicated rather than the mobilization of the sinus. Epilepsy due to inflammatory conditions in early childhood, such as meningitis, is not benefited in any way by the mobilization. In regard to idiopathic epilepsy I have found that about 1 case in 5 responds to this operative procedure.

OPERATION

For the alleviation of this condition, an operation has been devised. A cross-bow incision is made from mastoid to mastoid. The bleeding from the margins is controlled by Andrews clips. The lower flap is reflected downward to the attachment of the muscles at the superior nuchal line. These muscles are sectioned crosswise and immediately picked up in a Kellie forceps, and a suture is placed so as to draw the muscle margin firmly to the aponeurosis above. Next, the flap is reflected downward until the margin of the foramen magnum comes into view, and self-retaining retractors are placed. After the field has been draped with small towels, four trephine openings are made (Fig. 13), one on either side of the midline about equidistant from the superior longitudinal sinus and the transverse sinus, and two on either side of the midline below the transverse sinus. The two lower openings are then joined by rongeuring and the bone is removed down to and including the margin of the foramen (Fig. 14). With a DeVilbiss the two upper trephine openings, as well as the lower and upper on the same side, are joined. The bone is then gently released. Care must be used not to pull away roughly the venous attachment at the torcular Herophili. By the sliding of a small separator from below upward, as much of the emissary vein as possible is saved. Frequently, this can be ligated but if it should tear free from the sinus, a pledget of muscle will control the hæmorrhage promptly. If there is insufficient release of pressure within the first 3 or 4 minutes after the bone has been removed, the bone overlying the transverse sinus should be bitten away, well out into the mastoid area. A change in pulse pressure will be noted by the anæsthetist. The pulse will become quite thready and irregular, then return quickly to approximately the normal rate. The bone is gently replaced (Fig. 15) and the skin flap is replaced and closed by interrupted sutures, a single rubber band being inserted for drainage. A bandage is placed not too snugly over the area operated upon, but sufficiently tight to prevent oozing.

Patients are always restless and sometimes move their heads quickly when coming out of the anæsthetic; therefore a rubber band is inserted to take care of oozing. The dressings are removed

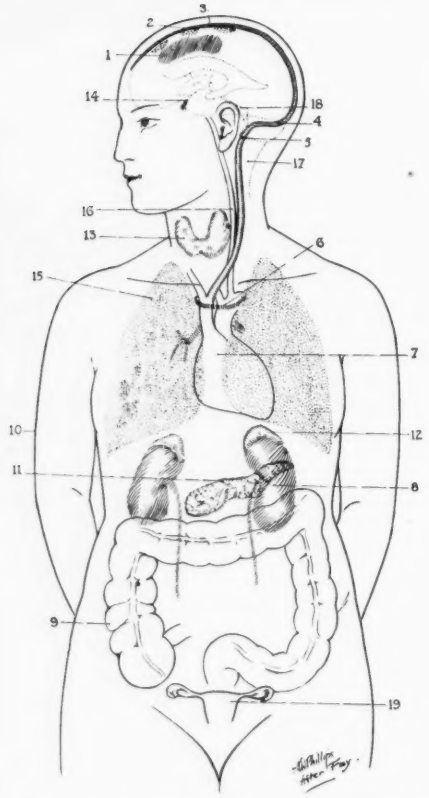

Fig. 8. Showing the points at which a lesion may occur which will cause accumulation of cerebrospinal fluid. *1*, Cortical lesions situated in the frontoparietal areas of the brain; *2*, the Pacchionian bodies; *3*, the longitudinal sinus; *4*, the lateral sinuses and the sigmoid; *5*, the bony openings of the jugular foramen; *6*, venous obstruction, due to an enlarged thymus gland; *7*, foreign bodies in the œsophagus; *8*, acute and chronic involvement of the kidney; *9*, the colon; *10*, skin elimination in its relation to fluid volume; *11*, pancreatic function in regard to insulin and carbohydrate metabolism; *12*, suprarenal lesions with insulin and carbohydrate metabolism undetermined; *13*, thyroid mechanical pressure on the jugular veins; *14*, pituitary interrelation with metabolism and other glandular structures; *15*, intrapulmonary structures and factors concerned with respirations; *16*, carotid circulation in terms of cerebral blood volume; *17*, medullary centers and their control of peripheral vasomotor responses; *18*, acute mastoiditis and parasinus abscess; *19*, disturbance of the pituitary and ovaries during the menstrual cycle. (After Fay.)

after the first 24 hours and the small rubber drain, inserted for oozing, is withdrawn. The patient remains in the hospital for a week following the operation.

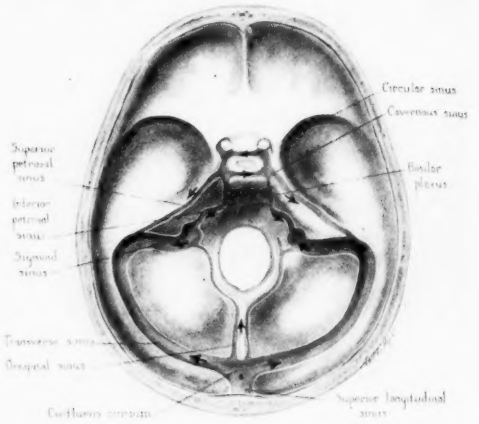

Fig. 9. Points of fixation of the transverse sinus are shown at the torcular where the emissary vein enters the sinus system. Emissaries enter also at the junction of the superior petrosal with the sigmoid on each side.

The danger points in the operation lie in hæmorrhage at the torcular Herophili and at the margin of the foramen magnum. Sometimes a branch of the vertebral vein will cause troublesome hæmorrhage. But aside from hæmorrhage there is no possibility of a fatality as the result of the operation. Convulsive seizures should not occur immediately after operation; however, if they do, the wound should be opened in search of a blood clot pressing upon the medulla. Convulsions due to pressure in this region are associated with sighing and more or less cyanosis.

As many of these patients have associated gastro-intestinal disturbances, marked constipation, or enlarged colon, it is a good plan to give a thorough catharsis several days before any operative procedure. If this is done, there will be no complications. Occasionally the adhesions between the dura and the bone over the cerebellar fossa are so firm that the dura and arachnoid are torn so that escape of cerebrospinal fluid, though normally of little consequence, may become quite extensive. If the dressings become rather moist and shock symptoms develop, it is well to give salt solution intravenously.

SUMMARY OF CASES

Series 1 includes the first 33 cases (Table I), operated upon from October 3, 1925 to November

Fig. 10. A large right transverse sinus which deviates from the falx cerebri before it reaches the fixation point at the torcular, thereby lending itself easily to compression with displacement downward of the tentorium. A left rudimentary sinus. Marked backward bulging of the dura overlying the left occipital lobe.

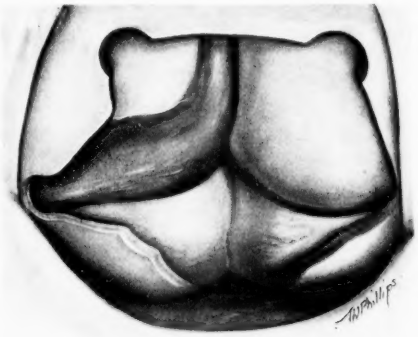

Fig. 11. A dilated superior longitudinal sinus draining into an enormously dilated left transverse sinus. There is a rudimentary right transverse sinus and a very small occipital sinus normally placed.

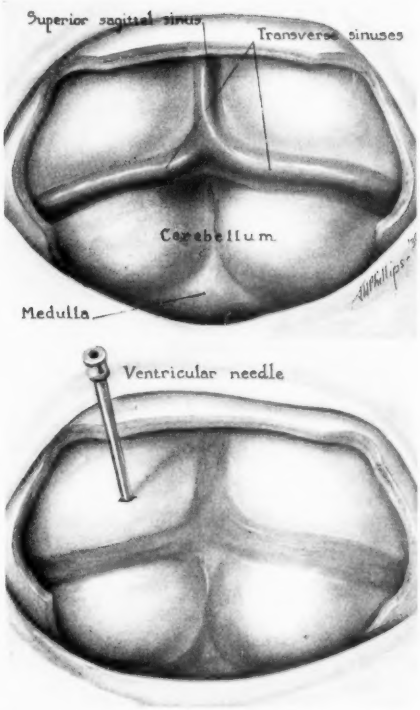

Fig. 12. Normal sinus system in which marked intracranial pressure causes uniform dilatation of the superior sagittal and transverse sinuses as shown above. Below is seen the result of release of pressure above the tentorium. Acoustic nerve tumor. P. W.

23, 1927, a 2 year period. The operation consisted of the removal of the bone overlying the medulla, extending from the posterior margin of the foramen magnum to the inferior curved line of the occipital bone, approximately 2 centimeters in width. There was one postoperative death due to hæmorrhage; one patient died 9 months after operation; another 3 months after operation. The average age of these patients was 10 years; the average pressure of the spinal fluid was 28 millimeters of mercury. In the patients under 10 years of age (16 cases), all of whom were given an anæsthetic, the average age was 3.8 years; the average pressure was 31.5 millimeters of mercury. In the degree of improvement in both grand and petit mal, there are very few (only 5 cases) showing marked (3+) improvement; 3 cases are cured.

These patients were all true epilepsies referred mostly from the Children's Orthopedic Hospital Clinic or from family physicians for operation. Toward the latter part of the list more and more bone was removed. Improvement was not uniformly present; therefore, it seemed that in Case 33 still more bone might be removed with safety. The marked improvement of this patient and the presence of large transverse sinuses prompted the extension of the operative area to include a decompression of the transverse sinuses on either side to the bend at their junction with the sigmoid and superior petrosal sinuses.

Series 2 includes 65 cases (Table II), with the more extensive operation in which the average age was 15 years; the average spinal fluid pressure was 29 millimeters of mercury. Those under 10 years of age given a general anæsthetic—19 cases—had an average age of 6 years, pressure being 27.4 millimeters of mercury. The average of both

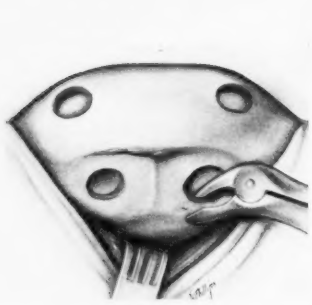

Fig. 13.

Fig. 14.

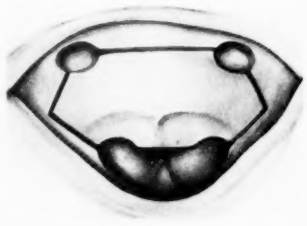

Fig. 15.

Fig. 13. Skin and muscle flap retracted downward to the margin of the foramen magnum. Trephine opening on either side of the midline above and below the transverse sinuses has been made. With the rongeur lower portion of the bone is removed.

Fig. 14. The bone has been removed over the cerebellar hemispheres and below to the foramen magnum. The margin of the foramen magnum has been removed for approximately 1 centimeter with the rongeur. The two upper trephine openings and the lower and upper on the same side are being connected with the DeVilbiss.

Fig. 15. The bone flap is replaced covering the torcular and transverse sinuses but leaving the enlarged foramen magnum uncovered.

series of cases shows 29 millimeters of mercury, while under anæsthetic the total (35 cases) showed an average of 4.9 years and an average pressure of 29.4 millimeters of mercury.

Mental deterioration was present in fewer cases, but it was more marked when present in this than in the former group. This was due to the fact that a more careful selection was made, since it had been found that mentality improved after operation only where deterioration was not too pronounced. In the presence of marked cerebral atrophy, as shown by air over the cortex and by marked mental deterioration, no improvement can be expected. Case 27 (Table II) shows marked mental deterioration with little air over the cortex (1+). Case 63 is similar to the above but has shown improved mentality.

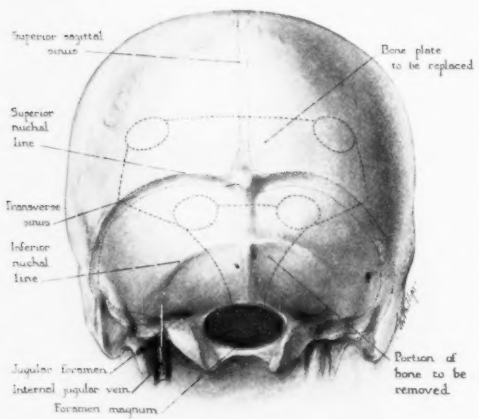

Fig. 16. Posterior view of skull showing the operative area.

In the early cases the air was injected and X-ray pictures were taken immediately, the reading of the encephalogram being based upon a 1+ to 4+ grade. Later (Table II), the dilatation was not considered so important as the cortical air; hence, this series is more carefully selected and shows a general marked improvement in grand mal attacks (25 cases) and in mentality (28 cases). While very little improvement is noted in petit mal attacks (6 cases), a total of 7 cases report no seizures since shortly after the operation.

FINAL OPERATION

The improved operation (Table II), while in a general way corresponding to the ordinary occipital or cerebellar decompression, exposed also the two transverse sinuses from just outside the torcular Herophili to the mastoid margin. The bone was removed entirely. The chief objection to this procedure was that the parents did not like to feel a soft spot on the back of the child's head. About this time, H. S. Souttar reported on the use of the electric craniotome in cerebellar operations, and he described mobilization of the torcular Herophili in his operative technique. This suggested the advisability of removal of a larger area of bone and replacement of the portion overlying

TABLE III.—GROUP 3—TWELVE CASES

Case	Age	Age at onset: Petit mal	Age at onset: Grand mal	Mental deterioration (−)	Encephalography: Date of examination	Encephalography: Pressure cerebrospinal fluid	Encephalography: Ventricular dilatation	Encephalography: Cortical air	Results of present operation: Date of operation	Degrees of improvement (+) in attacks and mentality: Petit mal	Degrees of improvement (+) in attacks and mentality: Grand mal	Degrees of improvement (+) in attacks and mentality: Mentality	Sinus anomalies
	yrs.	yrs.	yrs.			mm.							
1	10½		10		1–15–30	30		+	1–22–30		+++		Both large.
2	17		7		9– 5–29	22		+	1–27–30		++		Both large, occiput thick.
3	12	2½	2½	−−	2– 1–30	30	+	+	3–22–30	+	+++	+++	Left greater than right.
4	9	4	4	−	2– 1–30	40		+	4– 2–30	+	++	+	Occipital bone thick.
5	25	8	8	−	4–21–30	26		+	4–25–30	+	+++	+++	Left 3 times greater than right.
6	17	12	12	−	5– 1–30	22	+	+	5– 5–30	+		+	Right and left large, occiput thick.
7	22		21		5– 3–30	28		+	5– 7–30		+++	++	Left 2 times greater than right.
8	21		5	−−−	7– 7–30	28		+	7–17–30		+++	+++	Left 2 times greater than right.
9	20		13	−−−−	5–23–30	30	+	+	8– 1–30		+++	+++	Right 4 times left; left rudimentary.
10	16		12		6–10–30	28	+		8– 5–30		+++	+++	Right 4 times left; left small.
11	20		16	−	8–27–30	30	+		9– 1–30		+++	+++	Left 4 times greater than right.
12	25	Birth	24		2– 4–30	25	+		9– 9–30		+++	+	Right and left small, occipital large.
Average 18 yrs.					Average 28 mm.				Totals	4+	11+	10+	

TABLE IV.—A STUDY OF THE VENOUS SINUSES IN 70 PATIENTS SHOWS 57 HAVING ANOMALIES

	Cases	Per cent
Dilatation of both right and left transverse sinuses	13	18.5
Dilatation of right only	17	24.3
Dilatation of left only	9	13.
Contraction of right and left transverse sinuses	1	1.4
Contraction of right only	3	4.2
Contraction of left only	14	20.
Total	57	81.4

the sinuses (Fig. 16). This has proved entirely feasible and satisfactory.

Group III shows the final summary of 12 cases (Table III), carefully selected during the past year (1930). The average age is 18 years and the average pressure is 28 millimeters of mercury; 9 cases showed cortical air, while 6 showed dilated ventricles—3 showed both; 2 cases showed marked mental deterioration.

CONCLUSIONS

1. The periodic convulsive seizures associated with so called idiopathic epilepsy begin with a gradual accumulation of cerebrospinal fluid over the cortex and in the cisterns of the brain, causing a constant irritation to the cortex, particularly to the motor areas.

TABLE V.—A STUDY OF OCCIPITAL BONE CHANGES IN ONE HUNDRED AND TEN PATIENTS SHOWS EIGHTY-TWO HAVING VARIATIONS

	Cases	Per cent
Thickened	25	22.7
Thinned	27	24.5
Shortened	30	27.2
Total	82	74.4

2. The blocking of the cerebrospinal fluid may be due to inflammatory conditions of the arachnoid or Pacchionian bodies, or be produced by pressure upon any of the venous channels from the superior longitudinal sinus to the heart itself.

3. A general state of hydration is essential for the convulsive seizure in all instances.

4. Anomalous development of the venous dural sinuses is an important factor in epilepsy. Of these sinuses, the transverse sinus is of the most importance.

5. In those cases of anomalous development it is possible, by mobilization of the transverse sinus, to restore a sufficient venous return to prevent, under normal conditions, an accumulation of cerebrospinal fluid in the subarachnoid spaces.

6. The only complication that may arise during the course of this operation is postoperative hæmorrhage. This can be avoided by careful attention to all vessels at the time of operation.

7. Indications for this operation are the presence of anomalous venous sinuses in true epileptics with otherwise normal physical condition.

8. Contra-indications for this operation are the presence of disturbances due to inflammatory conditions, resulting in obliteration of the openings into the superior longitudinal sinus (Pacchionian bodies). Localized accumulations of cerebrospinal fluid over the cortex are, of course, not benefited by this operation.

9. The results of this mobilization bring about a marked improvement in the mental condition of the patient, as well as a decrease to a large proportion of the grand mal attacks and, to a less degree, the petit mal attacks.

10. The operation is applicable in only 20 per cent of cases.

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MARCHING FRACTURES OF METATARSAL BONES

With a Report of the Pathology

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INSIDIOUS fractures of metatarsal bones may occur after exhaustion of the normal muscle and tendon support to the foot. Such fractures occur without obvious trauma, and without sufficient immediate pain to notify the patient of the injury. Weeks later the developing callus becomes painful and calls attention to the lesion, but by this time the swelling is apt to be considered a neoplasm unless the observer is familiar with the picture of a marching fracture. Search of the literature shows that the problem has engaged several European authors, but no American and only one English article refers to it. The following case is reported in some detail as it is quite characteristic of the lesion. The patient had been previously seen by very good surgeons and roentgenologists who had said she was suffering from a sarcoma, and she had been sent to the hospital to have an amputation of the leg.

The patient was a big, healthy and rather obese woman of 30 years. She worked in the employees' cafeteria of a large company, and was on her feet all day long. She came to the hospital complaining of pain and a hard tumor in the left foot. Seven weeks previously she had noticed that she suffered pain just proximal to the second metatarsophalangeal joint when she bore weight upon the left foot. There was no trauma to the foot at this time as far as she could remember. The onset of the pain was very gradual, so that she could not remember just when she first noticed it; but she remembered it troubling her as she stood behind her counter about 7 weeks before. She did not stop work, but as she continued, the pain, which was slight at first, became gradually more severe. After about a week of this, the pain began to trouble her even when she was not on her feet, and at night she began to experience a steady aching pain in the same region. This, also, gradually increased so that shortly it began to keep her awake a good part of the night. She had lost about 13 pounds since the trouble began, and she attributed this to her loss of sleep as her appetite remained good and she had felt perfectly well all the time except for the pain in the foot. About 3 weeks after the onset she noticed that the dorsum of the foot seemed swollen, and at this time discovered that she could feel a hard lump about an inch proximal to the second metatarsophalangeal joint. The lump had slowly increased in size up to the time of her hospital admission. Two weeks before this date, she had reported to the medical department of the company employing her. She was referred to a very competent surgeon. X-rays were made. She was told she was suffering from a malignant tumor of the bone, and amputation of the leg was advised.

Eighteen years ago the left leg was run over by an automobile and the left ankle was sprained. This healed promptly and has never given further trouble. She had scarlet fever 18 months ago. Apart from these, her past history was negative. She was married and had two healthy children.

Examination at the time of her hospital admission was entirely negative with the exception of the left foot. She entered wearing a soft slipper on this foot, and although she walked readily when requested to do so, she walked with a decided limp. The dorsum of the foot was slightly œdematous and pitted on pressure. At the junction of the distal and middle thirds of the second metatarsal bone, a definite, very hard mass was felt. This was the size of a small walnut and gave very much the same sensation of a hard, irregular, globular surface as would a walnut felt under the skin. The skin was freely movable over it, but the mass was attached to and moved with the metatarsal bone. Pressure firmly made over the mass caused moderate pain. Voluntary and passive movements of the second toe caused pain. No abnormal mobility of the metatarsal and no crepitus were elicited. Routine blood and urine examinations were negative as was also the blood Wassermann test.

Roentgenograms (Fig. 1) showed a globular new-growth of bone at the site of, and slightly smaller than, the palpable tumor. This entirely surrounded the shaft of the bone. The margins of the growth were hazy and indistinct. The medulla of the metatarsal showed no changes. The medial surface of the cortex under the new bone showed a definite, irregular, shallow erosion. No fracture lines were made out in any of the four films examined, although as fracture callus was suspected, these were looked for carefully.

A luetic periosteitis seemed unlikely in view of the negative history and Wassermann report. An unrecognized fracture with excessive callus formation seemed unlikely in view of the insidious onset, the absence of corroborating X-ray evidence, and the destructive erosion of part of the cortex of the shaft beneath the new bone growth. A neoplasm that had involved a portion of the cortex seemed the most likely possibility. The metatarsal was excised as a means of verifying the diagnosis.

Examination of the specimen (Fig. 2) showed a bulbous enlargement 1.6 centimeters long by 1.7 centimeters wide at the junction of the distal and middle thirds of the shaft. The soft tissues were firmly adherent to this. The surface was rough, with small, quite regular elevations about 1 millimeter in diameter. It was quite hard but could be dented with the fingernail. There was no point of false motion discovered on first examination of the metatarsal, and it was only after longitudinal section of the bone that the true character of the lesion became evident. There was a narrow, dark line of old hæmorrhage and granulation tissue crossing the shaft almost transversely at the level of the bulbous enlargement. This stopped at the inner side of the callus on either side and did not involve the new-growth outside the cortex. When stress was applied to the bone there was a very slight abnormal mobility visible at this line, but the new bone callus surrounding it supplied such efficient splinting that the movement was slight and had been unnoticed before sectioning the bone.

Microscopic sections through the new bone growth showed it to be well developed and partially calcified osteoid tissue. There was no evidence of inflammatory reaction. The microscopic differentiation between young callus after fracture and neoplastic disease can be extremely difficult, but here there was nothing to suggest that the latter rather than the former was responsible for the picture. The lesion

Fig. 1. Anteroposterior and lateral roentgenograms of the foot.

was diagnosed as a fracture of the shaft of the bone with incomplete repair, small hæmorrhage and exuberant callus due to lack of immobilization of the fragments.

In 1855 Breithaupt described cases of persistently œdematous and painful feet occurring insidiously and without history of injury in soldiers who had been subjected to long marches, and gave this lesion the name *Fussgeschwulst*. He explained it as a traumatic inflammation of the tendon sheaths of the foot, and stated that several weeks of rest and abstinence from walking relieved the condition. Weisbach in 1877 explained the same lesion in soldiers as being due to traumatic inflammation of the intermetatarsal ligaments rather than the tendons, and gave it the name of *Syndesmitis Metatarsea*. Pauzat and Poulet wrote about the same process, but pointed out that it was primarily associated with marked periosteal proliferation (*Periostite Osteoplastique, Osteoperiostite Rheumatismale*). Later, aided by the advent of the roentgenogram, a large group of authors, among whom were Schulte, Kirschner, Momburg, and Baehr, began to recognize that in the majority of cases, despite the insidious onset and absence of definite severe trauma, there was a fracture of a metatarsal bone. Their large groups of cases were entirely among soldiers who had been subjected to long marches and severe exhaustion. The lesion was recognized as being quite common. Thiele reported 17 cases from one battalion in 3½ months. The association between the lesions and severe exhaustion of the patient's foot became clear, especially when the march was made with heavy pack and full equipment. It was intimated that the commanders rather than the feet were at fault; the rarity of the lesion in civilian practice was considered due to the civilian's proclivity to rest when he was tired. Momburg explained the fractures as the result of an inflammatory change in the bone due to prolonged elastic bending of its structure. This would first result in persistent proliferation, and if the stress continued, in a fracture. He radiographed the feet of soldiers who had no foot complaint and showed that silent periosteal proliferations of the second and third metatarsal bones were common. Kirschner stated that the lesions were all due to fracture or infraction of the shaft of the bone. This was often not recog-

nized at first because there was little or no displacement of the fragments and they were well immobilized by ligaments. The fractures occurred when the defense of the long toe tendons and the intrinsic muscles of the foot, which ordinarily protect the metatarsal against much of the stress of walking, was eliminated by exhaustion. Jansen, in 1920, stated the theory that a spastic flat foot developed first in these cases. The spasm of the interossei resulted in a lymph and a venous stasis, and this in turn caused first periosteal proliferation and later an internal rearrangement of bony architecture which weakened the metatarsal so that a pathological fracture resulted. He also had roentgenogram series showing at first only periosteal proliferation with fracture appearing later. The incidence of these fractures—54.5 per cent in the second metatarsal, 36.8 per cent in the third, and 2.5 per cent in the fourth—seemed to corroborate this as these are the metatarsals having bilateral interosseous muscle attachments; but the more simple, mechanical explanation of Kirschner—that is, exhaustion of muscle and tendon protection—is an equally satisfactory justification of this incidence.

Deutschlander, in 1925, described a group of 6 cases very similar to the one I have described. Instead of being soldiers, as in the previous cases, his patients were all women in the third decade. They developed pain over the distal shaft of the second or third metatarsal that gradually increased. There was no history of trauma. By the eighth or ninth week an encircling periosteal new bone growth was demonstrable in the roentgenogram. This persisted for some time and then gradually became more fusiform and smaller and the symptoms disappeared. Because 3 of the patients showed a low febrile course, because the callus took 8 to 9 weeks to become evident instead of the usual 3 to 4 weeks of fracture callus and because of the absence of evidence suggesting trauma, he believed his cases could best be explained as a low grade, hæmatogenous bacterial periosteitis. His stand is well taken and such a possibility cannot be controverted. However, he does not adequately explain the apparent immunity of the first and fifth metatarsal bones and none of Deutschlander's cases were examined after excision. In this, we have a case in which the sex, age, and clinical course simulate those of Deutschlander's so called "inflammatory tumor" and there was no history or roentgenographic evidence suggesting trauma. Yet examination of the metatarsal shows a concealed fracture, and histological examination of the callus shows no evidence of bacterial inflammation.

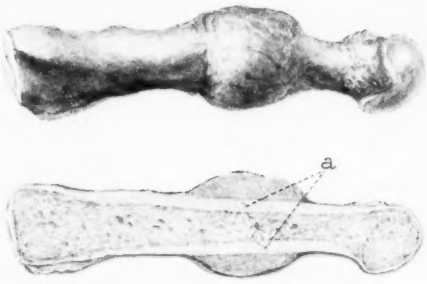

Fig. 2 Drawing showing line of fracture at *a*.

From a clinical standpoint, such cases should call forth the following reaction: When a case of apparent neoplasm involving the periosteum of the second, third, or fourth metatarsal bones presents itself, we should be most hesitant to rule out march foot. A luetic periosteitis may be excluded by the history and serological findings. Central neoplasms of the metatarsal shaft will be verified or excluded by the roentgenogram. But the roentgenogram is not to be relied upon too strongly in differentiating this type of fracture from a periosteal neoplasm. The negative evidence of fracture is misleading as is also the erosion of cortex under the new bone growth. Especially, if the callus is at the site of predilection of this type of fracture —at the junction of the distal and middle thirds of the second or third, more rarely the fourth (11) metatarsal—we should hesitate to consider neoplasm. Unless the case for neoplasm were incontrovertible we should put such patients to bed with the foot immobilized. In a relatively short time after this has been done, the pain will diminish and the march foot tumor begin to grow smaller.

The treatment of the cases should be elevation of the foot with immobilization for 2 or 3 weeks (2). Use of the foot actively may be started the fifth or sixth week. In the military cases, the average period of disability from duty was 49 days (12), but this should be somewhat shorter in civilian patients.

CONCLUSIONS

1. Fracture of metatarsal bones may occur insidiously as a result of long continued walking or standing.

2. Pain and callus formation later may simulate neoplasm.

3. It is suggested that some cases of Deutschlander's disease may belong in this group of fractures of metatarsal bones.

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OBSERVATIONS IN RAYNAUD'S DISEASE[1]

With Histopathologic Studies

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In 1862, Raynaud separated from the general grouping of "gangrene from arterial occlusion" a series of cases in which gangrene occurred without demonstrable organic disease or occlusion of the arteries. He ascribed the obstruction of blood flow in the arteries supplying the gangrenous part to an angiospasm of vasomotor origin. The chief diagnostic criteria laid down by Raynaud were as follows: (1) attacks of local syncope and asphyxia involving the fingers, hands, toes, and feet, more rarely the cheeks, nose, and lobes of the ears, the attacks usually being precipitated by temporary exposure to cold but occasionally from emotional excitement; (2) the local characteristic features of pallor, cyanosis, numbness, and lowering of surface temperature in the affected parts associated with slight discomfort, but occasionally severe pain; (3) in most cases a symmetrical distribution of the attack which lasts from a few minutes to several hours; (4) the tendency of the disease to be progressive—the local syncope, local asphyxia, and symmetrical gangrene being described as three successive stages of the same malady; (5) the presence of arterial pulsations in the affected parts except during the attacks of local syncope, when they may be feeble or absent; (6) the preponderance of females over males.

Nothing very important had been added to the clinical knowledge of this affection until recently when appreciation of the vascular changes occurring after certain operations on the sympathetic nervous system gave a rational treatment based upon the long accepted assumption that the disease is primarily vasomotor in origin. Leriche found that periarterial sympathectomy gave relief of symptoms in some cases of Raynaud's disease. Other observers (5 and 10), from animal and human experiments, have cast doubt upon the efficacy of periarterial sympathectomy in the treatment of vascular disorders of the extremities. Adson and Brown (1), while studying cases of spastic paralysis treated by lumbar ramisection, found a marked rise in the surface temperature of the desympathectomized limb, and appreciating the significance of this finding as applicable to the treatment of vascular disorders of the lower extremities, operated upon a patient with Raynaud's disease with strikingly beneficial results. Others (6) soon published similar results as regards the lower extremities. However, removal of the cervical sympathetic chain was not followed by complete or permanent relief of symptoms in the hands.

After Kuntz (9) showed that there is often a connection between the second thoracic sympathetic ganglion and the first thoracic spinal nerve, Adson and Brown (2) reported the successful treatment of a case of Raynaud's disease by resection of the inferior cervical, and the first and second thoracic sympathetic ganglia with the intervening trunks. In a later report, these same observers reported successful outcomes in 2 patients treated in the same manner. The results have been sufficiently encouraging to warrant the following statement by them: "The striking,

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maintained, and unequivocal therapeutic effects of lumbar and dorsal sympathetic ganglionectomy in Raynaud's disease seem to warrant the belief that surgical control of this disease is an accomplished fact."

The first cloud appeared on the horizon when Lewis took issue with the generally accepted belief that Raynaud's disease is due to vasomotor overactivity. His researches established reasonably well that a "local fault" in the vessel itself, rather than a nervous mechanism, was responsible for the symptoms of this disease. His conclusions were based upon indirect evidence, and no pathological studies were forthcoming to support his theories. A short time after his original report, Lewis (13) published observations on one postoperative case of Raynaud's disease. He found that he was able to induce typical attacks in the desympathectomized extremity (upper extremity) by exposure to cold. These observations confirmed his first conclusions and gave further weight to his theory of "local fault" predominating over vasomotor spasm as the causative agent of the disease.

This new point of view concerning the etiological factors in Raynaud's disease has cast considerable doubt on the theoretical soundness of sympathetic nerve surgery as a curative agent in this condition. The purpose of this paper is to review the details of our experiences with two severe cases and one moderately severe case of this disease treated by sympathetic ganglionectomy. Also the histopathological findings in the gangrenous fingers removed from one of the patients before and after sympathectomy are presented.

CASE REPORTS

CASE 1. H. G., sister of patient in Case 2, white, female, housewife, aged 24 years, first came under our observation in August, 1929. Six years ago she became extremely sensitive to cold weather, especially in the fingers and to a less degree in the toes. In December, 1928, after exposure to cold, she noticed that the ring finger of the left hand stayed numb and white. Soon discoloration appeared from the second joint to the tip of the finger, and in a few days it was completely black with a sharp line of demarcation at the level of the second phalangeal joint. She did not complain of pain. After the gangrenous finger had "shriveled" and become very hard and dry, it was amputated just proximal to the line of demarcation. Healing progressed satisfactorily. She continued to have attacks of pallor and numbness of the fingers and occasionally of the toes, brought on usually by exposure to cold but occasionally by emotional excitement. In August, 1929, the index finger of the right hand became gangrenous without warning (Fig. 1). She has had occasional attacks of local syncope in the right side of head, cheeks, and eyelids. The family history was negative for similar vasomotor disturbances, except for her only sister, who, likewise, suffers from Raynaud's disease, and her record is reported here as Case 2. The past history was irrelevant.

The general physical examination was negative except for the extremities. Blood pressure was 110-70. Laboratory examinations of urine and blood gave normal findings. Blood Wassermann was negative. Electrocardiograms and X-ray examinations of the heart and lungs were normal.

Extremities. Ring finger of left hand was missing. Index finger of right hand was gangrenous with a line of demarcation at middle of the first phalanx. The radial pulse was easily palpable. Surface temperature studies gave readings between 24 and 26 degrees C. (see Chart). Capillary circulation in finger nails was sluggish. Acetylcholine hydrobromide 100 milligrams given intramuscularly caused a rise of surface temperature to 36+ degrees C. Capillary circulation in the nail beds became active after the administration of this drug (see Chart).

First operation was done August 21, 1929. A bilateral cervicodorsal sympathetic ganglionectomy was performed. On the left side, the inferior cervical, first and second thoracic sympathetic ganglia with the intervening trunks were removed. On the right side, the stellate ganglion was broken during the process of removal, and the upper portion was not excised. Immediately following the operation, the upper extremities, neck, and face were warm and dry. Surface temperature readings of the hands ranged from 35 to 37 degrees C. A bilateral Horner's syndrome was present. The gangrenous finger improved steadily while the patient was in the hospital, the line of demarcation receding slowly until only the finger tip was involved (Fig. 2). She continued to have attacks of coldness of the feet and toes, and on one occasion the right little toe became cold, cyanosed, and numb.

She returned to the hospital on September 23, 1929, for lumbar sympathectomy. Her condition on the second admission was essentially the same except the Horner's syndrome was less complete. No sweating or reflex sweating was noted. The finger, which had been gangrenous, was now healed and of good color. Only the acra was lost, and a small portion of the nail had regenerated. No symptoms of Raynaud's disease referable to the hands had been noted.

On September 25, 1929, a bilateral lumbar sympathetic ganglionectomy was performed. The second, third and fourth lumbar sympathetic ganglia with the intervening trunks were removed by the transabdominal route. The lower extremities immediately became warm and dry. Skin temperature of the toes was constant at 36 degrees C. The patient complained severely of a dull aching sensation, deep in the legs and thighs, which was only temporarily relieved by elevation. This distressing complaint disappeared after 10 days.

Patient returned on January 10, 1930, because of gangrene of the right index finger. This digit, after assuming a normal state following the cervicodorsal sympathectomy, had given no symptoms for 3 months, and then gradually became gangrenous. The line of demarcation formed at the same level as before the operation. The finger was amputated at the metacarpophalangeal joint and the specimen decalcified for histological study. No further attacks of local syncope or asphyxia either in the hands or feet had been experienced.

On February 5, 1930, the first recurrent attack of pallor, numbness and loss of function occurred in the ring and little fingers of the right hand. Examination showed these two fingers to be pallid and hypæsthetic in their entirety. Capillary circulation did not appear unduly sluggish. Surface temperature readings were 34 degrees C. Injection of the ulnar nerve with novocain showed a rise of surface temperature of 2 degrees C., and acetylcholine intramuscularly gave a like rise in temperature. The Horner's syndrome was incomplete, there being moderate dilatation of the pupils, more marked on the right side. Inasmuch as

Fig. 1. Photograph of digits of right hand, Case 1, showing early dry gangrene of index finger before operation. Line of demarcation imperfectly shown at base of proximal phalanx.

only part of the stellate ganglion had been removed on the right side at the first operation, it was decided to excise the entire chain below the superior cervical ganglion. This operation completed the removal of the sympathetic ganglia with the intervening trunks from the superior cervical down to the third thoracic ganglion. The persistent attack of syncope of the ring and little fingers was promptly relieved following this procedure, and the digits returned to their normal color and function.

Three months later, these same two fingers suddenly became discolored, and superficial areas of gangrene appeared. This followed an emotional upset occasioned by the death of a close relative. Periarterial sympathectomy was performed on the third part of the axillary artery. The circulation proximal to the gangrene improved following this procedure. The superficial devitalized tissue sloughed and again the fingers returned to a normal appearance. No further attacks were experienced until about 3 months later when again both fingers became completely gangrenous without any known exciting cause. Amputations

Fig. 3. Photograph of both hands, Case 2, before operation. The middle finger of the right hand was removed because of dry gangrene 7 months previously.

Fig. 2. Photograph of both hands, Case 1, showing the formerly gangrenous index finger 2 weeks after operation. The line of demarcation has receded to the middle of the terminal phalanx. The tip was subsequently removed and healing progressed normally. The missing ring finger of left hand was lost from dry gangrene 8 months previously.

were performed at the metacarpophalangeal joints, and the specimens prepared for histological study. At this time, the ulnar artery could not be palpated, although the radial artery pulsations were readily felt.

Two and one-half months later, the remaining fingers of the right hand (thumb and middle) went into a constant state of asphyxia and soon became black and gangrenous. Amputations were performed on November 8, 1930, leaving the patient devoid of digits on the right hand. The last fingers were so completely decomposed that no histological sections could be satisfactorily prepared.

Case 2. L. S., Female, white, housewife, aged 22 years and sister to H. G. of Case 1. The onset of her symptoms antedated those of her sister by one month. In 1924, she first noticed that her feet and hands perspired freely without apparent cause. At the onset of winter of the same year, she thought that her hands and feet were more sensitive to the cold than usual. Her symptoms gradually progressed. Mild attacks of pallor, cyanosis, and numbness occurred in the digits of the right hand first, but the other extremities soon became involved in the attacks to a lesser degree. The attacks became more frequent and severe, and the remissions less complete.

In January, 1929, the middle finger of the right hand became cold, numb, and painful. Dry gangrene slowly developed with a line of demarcation at the distal end of the proximal phalanx. In March, 1929, this finger was amputated. The stump healed quickly and completely (Fig. 3). The attacks continued in the other digits, particularly in the great toe of the right foot. During the next 4 months, this toe slowly approached the pregangrenous state. She was first seen by us at this time.

Past history was essentially negative. She was nervous but not neurotic or hypochondriacal. Coffee was used in large quantities as well as cigarettes to the extent of one to two packages daily.

The physical examination revealed nothing of importance except for the extremities. Blood pressure was 102–60. The usual laboratory procedures revealed a negative blood Wassermann, slight secondary anæmia, normal electrocardiographic tracings, and a normal size heart as determined by roentgenographic studies.

Extremities. The middle finger of the right hand had been amputated at the junction of the upper and middle third of the proximal phalanx. The soft parts were healthy, and there was no evidence of delayed healing. At a room temperature of 26 degrees C., the extremities were moist

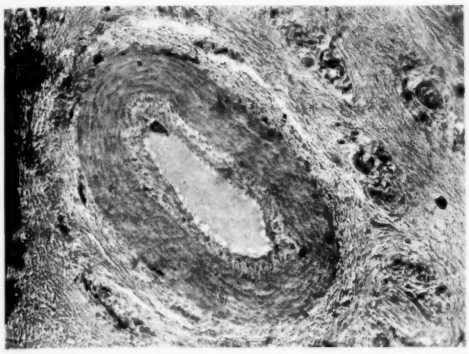

Fig. 4. Section of digital artery from middle phalanx of the index finger of right hand, showing thickening of the intermediary layer of the intima. × 70.

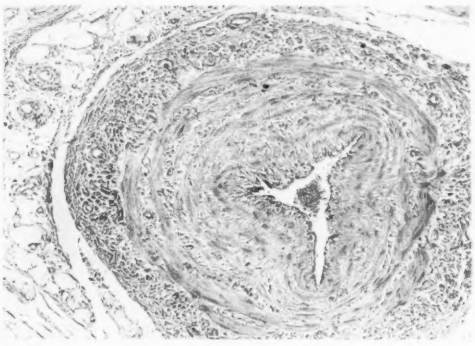

Fig. 5. Section of digital artery from proximal phalanx of the little finger of right hand, showing thickening of intermediary layer of intima, thickening of the media, with hypertrophy and separation of smooth muscle cells. × 70.

and felt cold with an actual reading of 29.2 degrees C. Surface temperature ranged as low as 24 degrees C. Acetylcholine, 100 milligrams intramuscularly, caused an elevation of surface temperature in all the extremities to 35+ degrees C. on repeated occasions. The radial pulse and the dorsalis pedis pulse were palpable bilaterally. The capillary circulation of the finger and toe nails was sluggish.

The first operation was performed September 25, 1929. The second, third, and fourth lumbar sympathetic ganglia with the intervening sympathetic trunks were removed bilaterally. The convalescence was uneventful except for an attack of vomiting and epigastric distention shortly after the patient was discharged from the hospital. After operation the lower extremities were warm and dry. The skin temperature over a period of 2 weeks ranged between 34 and 36 degrees C. There have been no further attacks of syncope. Patient has been free of all symptoms in her lower extremities up to the present date.

On October 10, 1929, the second operation—a cervicodorsal sympathectomy—was performed. The inferior cervical, first and second thoracic ganglia with the intervening sympathetic trunks were removed bilaterally. All symptoms subsided completely. The upper extremities were warm and dry. They were normal subjectively. A typical Horner's syndrome was present bilaterally. On February 8, 1930, the pupillary response to light was fairly good. The Horner's syndrome was incomplete. The patient perspired freely over the entire body but less than usual over the extremities. She has had no further recurrence of any symptoms 1 year and 4 months after the first operation.

Case 3. M. M., female, white, aged 9 years, referred by Dr. Tom Marks of Lexington, Kentucky, was first seen by us on June 15, 1930, with the complaint of blanching and numbness in the fingers of both hands and occasionally similar attacks in both feet. In January, 1930, while coming from Sunday School, she noticed a cold, numb feeling in the tips of the fingers of both hands, and within a very short time, this feeling had progressed upward to the elbows. The mother states that the hands were pallid at this time. From January, 1930 to June 15, 1930, several attacks in the upper extremities had occurred. These attacks were characterized by blanching and then cyanosis, accompanied by numbness and a slight degree of pain. Only occasionally would more than the fingers be involved. Four was the largest number of attacks recorded in 1 day. She noticed that her chin and left cheek became pale and numb on two occasions for a period of 10 to 15 minutes. The lower extremities had suffered six light attacks prior to June, 1930. The family and past histories were irrelevant.

Patient was a well developed, well nourished, and very alert child, who was mentally quite precocious. Physical findings were not remarkable, except for the extremities. A definite cyanosis was noticeable. Pulsation of radial and dorsalis pedis arteries was plainly perceptible bilaterally. Capillary circulation was very sluggish in finger and toe nails. Surface temperature ranged between 25 and 29 degrees C. during the period of observation. Room temperature at this time was 24 to 26 degrees C. Acetylcholine hydrobromide, 50 milligrams intramuscularly, increased the surface temperature by 7 degrees C. Attacks could be induced by immersing the hands in water at 15 degrees C. for 15 minutes. On a particularly warm day, it was impossible to induce an attack in this manner.

On June 23, 1930, the inferior cervical, the first and second dorsal ganglia with the intervening sympathetic trunks were removed. Convalescence was normal. Both upper extremities became warm and dry. A complete Horner's syndrome was present. Patient was free of all attacks until the onset of fall weather 4 months later, when attacks in the lower extremities became more pronounced and occurred every 2 to 3 days. At this time, the little and ring fingers of the right hand also suffered repeated attacks, but less severe than before. Temperature of the upper extremities was 34+ degrees C. and only 29 degrees C. for the lower extremities with a room temperature of 22 degrees C. Typical attacks could be induced by immersion of the extremities to the wrists and ankles in water at 15 degrees C. for 15 minutes.

On October 29, 1930, the second operation—a periarterial sympathectomy was performed on the third part of the right axillary artery. The surface temperature was elevated 0.5 degrees C. after the operation, the temperature being 34.5 degrees C. with a room temperature of 22 degrees C.

Since the attacks in the lower extremities occurred more frequently and were more severe, third operation—a bilateral lumbar sympathectomy—was performed, November 18, 1930. The second, third, and fourth ganglia with the intervening trunks were removed on the right side. Only the fourth lumbar ganglion was removed on the left

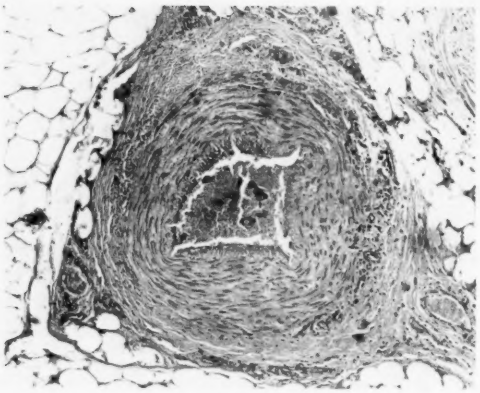

Fig. 6. Section of digital artery from distal portion of proximal phalanx of the little finger of right hand, showing thickening of the intermediary layer of the intima, thickening of the media with hypertrophy and separation of the smooth muscle cells. × 70.

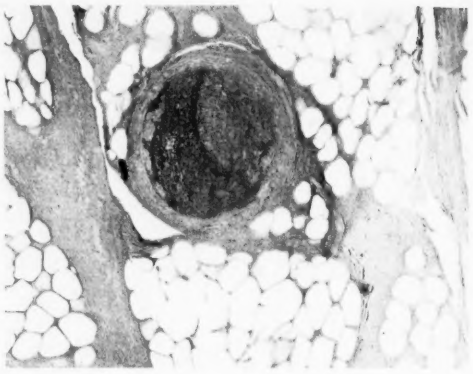

Fig. 7. Section of small artery from distal portion of proximal phalanx of little finger of right hand, showing obliteration of the lumen by thickening of the intima and a thrombotic mass. × 70.

side. Convalescence was uneventful. No further attacks have occurred in these extremities.

Communication of February 5, 1931, states that the patient has had one attack of syncope involving only the index finger of the right hand. This attack followed exposure to cold. No symptoms referable to the disease have been noted in the lower extremities and left upper extremity.

THE HISTOPATHOLOGY OF THE BLOOD VESSELS OF THE FINGERS IN RAYNAUD'S DISEASE BY JAMES B. ROGERS, M.D.

The histological evidence presented in the literature concerning Raynaud's disease is vague. Grenet and Isaac Georges reported lesions, inflammatory in character, involving the arterioles in Raynaud's disease. In 2 cases, lues was present, and in a third case, a mild infection was present. Simpson, Brown, and Adson (12) considered the mild case a vasomotor neurosis, and that abnormality of the digital arteries is a late complication.

The material presented here was taken from gangrenous fingers amputated from the same patient. The ring finger of the left hand was amputated in February, 1929; the index finger of the right hand in January, 1930; and the ring and little fingers of the right hand in August, 1930. Serial sections were cut at three levels in each finger.

OBSERVATIONS

Section through the middle phalanx of the index finger of the right hand. This portion was gangrenous and sections showed numerous areas of necrosis. The digital artery was somewhat flattened, and the lumen was filled with an amorphous débris. The endothelium was intact. The intermediary layer was much thickened, but not uniformly. The internal elastic membrane could not be seen. The intima showed few folds. The increase in thickness was due to an increase in connective tissue. The media was not thickened, and the smooth muscle cells had a normal appearance. The adventitia was normal (Fig. 4). The digital vein was filled with red corpuscles, the outlines of which were indistinct. A portion of the intima was adherent to the mass of corpuscles. The rest of the vein wall was normal. The lumen of small arteries was filled with blue staining cells. The endothelium could not be seen, although the intima was thickened and was not sharply marked off from the media. The media was not thickened. The lumen of small veins was filled with leucocytes. The endothelium was distinct. The rest of the vein wall was normal in appearance. In a small artery, which was 30 microns in diameter from adventitia to adventitia, the endothelial cell nuclei could be seen. The intima was in folds and the lumen obliterated. The intima made a circle about 10 microns in diameter. Capillaries were not distinct.

Section through the proximal phalanx of the little finger of the right hand. There was no external evidence of gangrene, and sections showed no areas of necrosis. The lumen of the digital artery was diminished in size and rather triangular in outline. The central portion was occupied but not filled by a mass of red corpuscles. The endothelium was broken, but some endothelial cell nuclei could be seen. The intermediary layer was thickened. There were numerous open spaces in this layer. The internal elastic membrane could not be seen. There was a marked separation of the layers of smooth muscle. There were open spaces, bridged by connective tissue fibers quite irregular in arrangement. The smooth muscle cells were hypertrophic and well stained. The external elastic membrane could not be seen. These changes could be found in the branches of the digital artery (Fig. 5). The digital vein was collapsed and the lumen empty. The endothelium was intact. The intima contained many open spaces and was not sharply marked off from the media. The media consisted of two or three layers of hypertrophic smooth muscle. The adventitia was clearly marked off from the media. The lumen of the small veins was filled with red corpuscles, and the walls presented a normal appearance. The lumen (10 microns in diameter) of an arteriole had a red corpuscle in it. The endothelium was

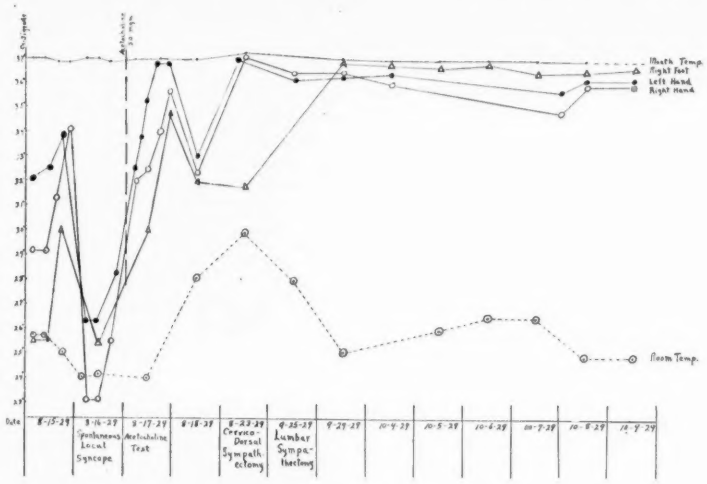

Chart showing temperature in Case 1.

intact. One layer of hypertrophic smooth muscle constituted the media. The endothelium in vessels (7 microns in diameter) was intact. Some of these capillaries were empty; others were blocked by red corpuscles.

Section through the distal portion of the proximal phalanx of the little finger of the right hand. There was no external evidence of gangrene, and sections showed no areas of necrosis. The lumen of the digital artery was rather triangular in outline and was filled with red corpuscles and a few leucocytes. There were some desquamated endothelial cells. Most of the endothelium was intact. Some portions of the intermediary layer of the intima were thickened. The internal elastic membrane could be seen. The media was not of uniform thickness. Where there was increase in thickness, there was separation of the muscle layers by open spaces. The smooth muscle cells were hypertrophic. The external elastic membrane could not be seen (Fig. 6). The digital vein was filled with a granular mass in which numerous red corpuscles could be seen. The endothelium could not be seen. The cells in the media stained poorly. The lumen of a small artery (Fig. 7) was completely obliterated by a thrombotic mass and a thickened intima. The endothelium could not be seen. The intima had no characteristic markings and was not sharply marked off from the media. The cells of the media stained poorly. Small veins were filled with red corpuscles. The endothelium was intact. The cells in the media and adventitia stained poorly. There was a great deal of hæmorrhage in this region. The lumen of an arteriole (12 microns in diameter) contained red corpuscles. The endothelium was intact. The smooth muscle cells of the media were hypertrophic. Vessels (7 microns in diameter) showed well stained endothelium. Most of the capillaries were blocked by red corpuscles. Other histopathological changes which were seen in the entire series of sections were listed in Table I.

Summary. 1. There were pathological changes in the blood vessels proximal to and in the areas of gangrene.

2. These changes were most evident in the digital arteries and their branches.

3. These changes were thickening of the intima, thickening of the media with hypertrophy of smooth muscle fibers, and separation of the layers of smooth muscles by open spaces.

4. There was obliteration of small arteries proximal to the areas of gangrene.

5. The media of arterioles contained hypertrophic smooth muscle cells.

6. The terminal portions of the fingers showed extensive coagulation necrosis.

7. The changes were more pronounced in the fingers amputated last.

8. The histologic evidence is summarized in Table I.

These cases we believe to be typical examples of Raynaud's disease; two severe and one moderately severe. All three have suffered from bilateral, but not symmetrical, progressive circulatory changes manifested by intermittent attacks of numbness, pallor, cyanosis, and lowered surface temperature, involving primarily the upper extremities and to a lesser degree the lower extremities. Two of them have progressed to gangrene of the fingers, and the other has had nutritional changes in the soft tissues of the hand and fingers. In one case, the attacks were limited to the fingers and toes. In the 2 other cases, the involved areas included the hands, forearms to the elbows, and occasionally the face, chin, and eyelids. Exposure to cold was the chief exciting agent, although in 2 cases, emotional excitement often precipitated attacks.

In each instance, the attacks were bilateral but not symmetrical, i.e. the same finger on each hand or the same toe on each foot. Pain was not a predominating feature of the disease; in 2 cases, the pain or discomfort was very slight, and in the

TABLE I.—SUMMARY OF HISTOPATHOLOGICAL AND CLINICAL DATA IN CASE I

Finger	Dates of gangrene	Operative procedures	Date of amputation	Section	Necrosis	Arterial changes	Venous changes
Ring finger left hand	12–1928	None	2–1929	Proximal	No	No increase in connective tissue	No changes
				Middle phalanx	Yes	Intima and media increased	No changes
				Terminal phalanx	Yes	Thickened intima; thickened media	No changes other than those associated with gangrene
Index finger right hand	8–1929	Cervicodorsal sympathectomy 8–21–29	1–10–30	Proximal	No	Intermediary layer thickened in areas	No changes
				Middle phalanx	Yes	Intermediary layer thickened in areas. Increase in connective tissue. Media not thickened	No changes
				Distal end	Less than middle	Thickened intima and media	No changes
Little finger right hand	1–1930	Stellate 2–5–30 Periarterial sympathectomy 5–19–30	8–11–30	Proximal	No	Lumen small. Intermediary layer thickened. Internal elastic membrane not seen. Media thickened and hypertrophied	Open spaces in media
				Distal portion of proximal phalanx	No	Portions of intima thickened. Internal elastic membrane visible. Media thickened in places	No changes
				Distal portion of middle phalanx	Yes	Lumen small irregular. Thrombotic mass. No endothelium. Intermediary layer thickened. Internal elastic membrane visible. Media thickened	Thrombotic mass
Ring finger right hand	Same as little finger	Same as above	8–11–30	Proximal portion of middle phalanx	No	Endothelium mostly destroyed. Intermediary layer thickened. Internal elastic membrane visible. Media thickened Muscles hypertrophied	No changes
				Proximal distal phalanx	No	Lumen small irregular. Intima thickened. Media thickened	Leucocytic infiltration

third case of only moderate severity. The discomfort was most severe as the attack was ushered in or departing. Normal arterial pulsations were observed in all 3 cases with the exception of the ulnar artery in Case 1 after all the digits had been lost from the right hand. All 3 patients were healthy individuals, except for this local disease.

The operative indications were clear cut in each case. All of them gave excellent vasomotor responses with the acetylcholine test; each patient experienced a rise of surface temperature in the involved parts to normal levels; (a rise of 12 degrees C. was not uncommon). Also the capillary circulation improved with each diagnostic dose of the drug.

Symptoms have been completely relieved in the lower extremities in 2 cases for 18 months and in the third case for 5 months following the operative procedures. In all of the patients, symptoms have been completely relieved in the left hand. Two cases have had recurrence of symptoms in the right hand; 1 of the 2 has progressed to gangrene of all the digits of this extremity, following a very extensive resection of the regional sympathetic system.

The conclusion that certain cases of Raynaud's disease of the hands are not curable or controllable by the present day operative measures on the sympathetic nervous system is apparent. Three possible explanations for this failure should have consideration: (1) local changes in the vessels, which may be the primary or secondary cause of the disease; (2) incomplete removal of the central connection of the vasomotor system to the upper right extremity; (3) the presence of a secondary vasomotor mechanism which is capable of functioning after all central communications are severed.

Local fault. Lewis' contention that the symptoms arising from this disease are due to local changes in the walls of the arteries would at first thought be borne out by the results obtained in these cases from sympathetic nerve surgery. (He does not attempt to say what this "local fault" may be nor from where it arises.) Such a contention would be further strengthened by the pathological changes observed in the fingers obtained from Case 1. Our only direct evidence of local arterial changes was obtained from the study of amputated fingers which had undergone re-

peated periods of prolonged ischæmia. In three of the four specimens, a fairly generous area of tissue proximal to the line of demarcation was available for study. In all specimens, the digital arteries and their branches showed definite evidence of disease, consisting chiefly of thickening and fibrosis of the intima with hypertrophy of the media. These changes were more pronounced in the gangrenous than in the pregangrenous portions. In attempting to interpret the significance of these changes, one must first consider the possibility of these arterial changes being the result rather than the cause of the gangrenous process. It is, we believe, a generally accepted fact that arteries imbedded in inflamed tissue become involved, and all their layers undergo fibrous thickening, especially the intima. If the changes we observed in the vessels were confined to areas which had been gangrenous, we would be inclined to attribute them to a reaction secondary to the gangrenous process. But very definite evidence of arterial changes was found proximal to the line of demarcation in tissues which were essentially microscopically normal in appearance.

The first significant fact obtained from the histological study of the arteries was the predominance of changes in the large digital arteries with relatively few changes in the arterioles. If the underlying pathology in Raynaud's disease is a local change, one would expect the smaller branches to be involved to a greater degree than was evidenced.

Another possible explanation of the observed pathological changes would be that the vessels became diseased as the result of the long standing attacks of angiospasm. With each attack, the vessels suffered locally, and as the changes in the vessels became more pronounced, their ability to function between attacks became more and more impaired. Finally, the end point of adequate blood supply was reached, due to angiospasm and arterial occlusion, and the part became gangrenous. The fact that more pronounced arterial changes were observed in the fingers which were amputated last would support this view.

Incomplete removal of the central sympathetic connection. So far as we are aware, there is no case of Raynaud's disease of the lower extremities on record which has not been completely relieved of symptoms following sympathetic ganglionectomy. This fact immediately suggests the possibility that the difficulties encountered in treating the upper extremities are due to an incomplete removal of vasomotor activity to these members. Based upon our present anatomical knowledge of the vasomotor supply to the upper extremities, every possibility of a central connection of these fibers in the upper extremities has been removed in Case 1, yet this patient has had no permanent clinical benefit from the sympathectomy on the right side. That there still may be vasomotor pathways to the arm of which we have no anatomical knowledge is not beyond the range of possibility.

It is known that many of the white rami do not end in the lateral ganglia of the sympathetic chain but proceed directly to various collateral ganglia. These ganglia act in many respects as a switchboard. One preganglionic fiber may synapse with many postganglionic cells. This permits of a diffuse and widespread action. The extensive communication of these postganglionic fibers may afford a long circuitous route through which vasoconstrictor impulses coming from a lower central level, namely, the third thoracic ganglion, may reach the blood vessels of the upper extremities.

There are more collateral ganglia in the region of the upper extremities, and a greater possibility for the dissemination of sympathetic impulses is thereby afforded, than in the case of the lower extremities.

Secondary vasomotor system. Another possible means of a continued sympathetic vasomotor action in the upper extremities is from a secondary system as described by W. Braeucker. He considers the sympathetic nervous system as an intercommunicating network of fibers. The blood vessels are supplied by a network of sympathetic fibers, which make central connections by a direct and indirect system. The direct system is a continuation of the sympathetic network on to the vessels, while the indirect system is transmitted by way of the rami communicantes to the spinal nerves. This network is a special apparatus and is capable of living and of continuing function independent of central connection. Normally, the vasomotor impulses are conveyed via the rami communicantes and the sympathetic nerves, but when this is interrupted, the auxiliary system acts as a substitute and transmits the impulses directly. So Braeucker presents a dual mechanism for the transmission of impulses as well as a network of sympathetic fibers and cells capable of functioning independent of central connection.

If his idea of the vasomotor system of the upper extremities is correct, the operative procedures as now practiced will not permanently interrupt vasoconstrictor impulses. This possibility should have consideration, but until further evidence is forthcoming, it should have less consideration than the former two.

While our evidence is insufficient to establish proof of the underlying mechanism of Raynaud's disease, we favor the assumption of overactivity of the vasomotor system as being the primary causative factor. Furthermore, we attribute the failure of receiving permanent relief of symptoms in the right upper extremity as being due in the early cases to an incomplete removal of the vasomotor mechanism. The arterial changes observed may result from, rather than being the causative factor of, the angiospasm. Obviously, in advanced cases where arterial changes have occurred, removal of the entire sympathetic mechanism still would fail permanently to relieve the symptoms.

It would seem most likely that the remaining vasomotor control, after the conventional cervicodorsal sympathectomy, must come from a lower segmental level. Accordingly, it is our intention to include the third thoracic ganglion in the next operative procedure for this disease.

PHYSIOLOGICAL DISTURBANCES AFTER SYMPATHECTOMY

1. Horner's syndrome. This phenomenon in all our cases has been observed to be complete for only a short period after removal of the first and second dorsal and inferior cervical ganglia with the intervening trunks. Usually, after 4 to 6 weeks, pupillary response has returned to a moderate degree. The sweating mechanism to the face and neck has apparently been permanently destroyed in 2 cases, but has partly returned in the third. Narrowing of the palpebral fissure has been insignificant in each instance when the ganglia mentioned were removed. In 1 patient (Case 1), in whom the middle, inferior cervical, first and second dorsal ganglia were removed unilaterally, a complete and permanent Horner's syndrome on that side was produced.

2. Cardiac studies after sympathectomy. In 2 of our cases, studies were made to determine the effect of cervicodorsal sympathectomy upon the accelerator mechanism of the heart. The response to graded exercise was studied both before and after operation, the pulse rate, blood pressure, and subjective symptoms being recorded.

No significant changes in the blood pressure were observed after cervicodorsal and lumbar sympathectomy. The response to graded exercise was normal after 4 weeks; the heart beat accelerated to the pre-operative level with all grades of exercise. The return to the pre-exercise rate was accomplished in the normal manner as determined for each patient. No cardiac distress or other symptoms of discomfort relevant to the cardiac mechanism were complained of even after fairly strenuous climbing of stairs.

Electrocardiographic studies showed no significant changes. Repeated X-ray studies of the heart over a period of 6 months following sympathectomy revealed no appreciable change in the size of the heart or aortic shadows. This latter observation came as a surprise to us in view of the cardiac output determination (8) made on Case 1 and Case 2:

Case 1. August 19, 1929—before operation:

Cardiac output	7.38 liters per minute
Mean circulation time	18.8 seconds
Vital capacity	3.35 liters
Amount of blood in heart and lungs	2.33 liters
	Pulse 70

Case 1. September 24, 1929—after cervicodorsal sympathectomy:

Cardiac output	5.88 liters per minute
Mean circulation time	19.8 seconds
Vital capacity	3.35 liters
Amount of blood in heart and lungs	1.94 liters
	Pulse 66

Case 1. October 8, 1929—after cervicodorsal and lumbar sympathectomy:

Cardiac output	3.95 liters per minute
Mean circulation time	19.0 seconds
Vital capacity	3.35 liters
Amount of blood in heart and lungs	1.25 liters
	Pulse 80

Case 2. September 24, 1929—before operation:

Cardiac output	6.62 liters per minute
Mean circulation time	17.4 seconds
Vital capacity	3.40 liters
Amount of blood in heart and lungs	1.92 liters
	Pulse 90

Case 2. October 9, 1929—after cervicodorsal sympathectomy:

Cardiac output	5.56 liters per minute
Mean circulation time	14.8 seconds
Vital capacity	3.40 liters
Amount of blood in heart and lungs	1.37 liters
	Pulse 104

Each output determination was made under as nearly identical conditions as possible. During the first experiment (Case 1), a state of moderate vasoconstrictor spasm was maintained, but, otherwise, the patient was composed and unexcited. The pulse rate during each experiment was practically constant. Unfortunately, it was impossible to make simultaneous cardiac output records and cardiac roentgenograms.

What significance to attach to the output determinations in these cases, we are unprepared to say. The possibility suggests itself in relation to the increased output reported under the action of adrenalin (14), that an increased cardiac load

is being carried in Raynaud's disease, which is relieved by sympathectomy. The mechanism of this diminution of output is inexplainable to us at the present time.

3. *Changes in libido, menstruation, etc.* One of these patients (Case 1) states that after lumbar sympathectomy, her previous frigid state became promptly altered to one of almost abnormal libido. This statement is corroborated by her husband. The other adult patient experienced no change in libido following her lumbar sympathectomy. She had experienced, however, normal sexual intercourse throughout her married life. We have observed in certain young male patients, treated for deforming arthritis by lumbar sympathectomy, a fairly constant semi-erection of the penis. Presumably, these effects upon the genital organs arise from hyperæmia of the parts resulting from the vasodilatation. This observation is in direct contrast to the observations on animals made by Bacq, in which he reports impotence following a more extensive removal of the regional sympathetic system.

The character and frequency of the menstrual cycle was unchanged in our patients.

In one instance (Case 1), the convalescence was complicated by severe burning, aching pains in the thighs, legs, and feet. The only relief found for this symptom was elevation of the legs above the level of the body and head. Drugs and cold applications gave little or no relief. This symptom gradually subsided and disappeared after 10 days. Presumably, it was due to distention of the vessels much like the symptoms produced by erythromelalgia.

SUMMARY

1. Observations upon 3 typical cases of Raynaud's disease treated by sympathetic ganglionectomy are reported.

2. Complete relief of symptoms in all cases was obtained in the lower extremities and left upper extremity for periods ranging from 6 months to 18 months.

3. The disease has not been permanently relieved in the right upper extremity of 2 cases; in 1 case, all the digits of the right hand have been lost by dry gangrene after removal of the entire sympathetic chain from the level of the superior cervical ganglion to the third thoracic ganglion, also after periarterial sympathectomy of the axillary artery.

4. Histopathological studies were made on the amputated digits. Sclerotic and hypertrophic changes were found in the intima and media of the larger arteries both proximally and distally to the line of demarcation. No significant changes were found in the arterioles or veins.

5. These clinical and pathological findings and their significance as regards the causative mechanism of the disease are discussed.

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ANOMALY OF RIGHT RECURRENT LARYNGEAL NERVE

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IN the performance recently of subtotal thyroidectomy on a woman with exophthalmic goiter, an interesting anomaly of the right recurrent laryngeal nerve was encountered. Since we had not previously observed such anomaly and since it is of surgical significance it seemed worthy of reporting. Because of the unusual and unexpected situation of this nerve it may easily be injured during resection of the right lobe of the thyroid gland.

In this case after resecting both lobes of the thyroid gland, we were intending to ligate the right inferior thyroid artery outside the gland and as close to the carotid sheath as possible, as it is often our practice as an additional measure in the prevention of postoperative hæmorrhage. A structure was encountered having the superficial appearance of the vessel and arising in about the right situation for the inferior thyroid artery. A ligature had actually been passed around the structure, but had not been tied, before more careful examination showed it to be not a vessel but a nerve. As a nerve had never been found in this situation, we were much interested in carefully dissecting it out in an effort to determine its identity. It was readily traced upward and was found to arise from the cervical trunk of the vagus at a point about opposite the superior pole of the thyroid gland. It coursed downward along the mesial border of the parent trunk for a distance of 3 to 5 centimeters, and then emerged from the carotid sheath, nearly at a right angle, to pass directly to the groove between the trachea and the œsophagus in the region of the inferior pole of the thyroid gland (Fig. 1). From here its ascending course apparently corresponded with that of the normal inferior laryngeal nerve. Further dissection revealed absence of any nerve along the lateral wall of the trachea below the level of the inferior pole of the gland. Therefore, the nerve first observed and so narrowly escaping injury was the only inferior laryngeal nerve on the right side. The left inferior laryngeal nerve appeared to be perfectly normal in its origin and course.

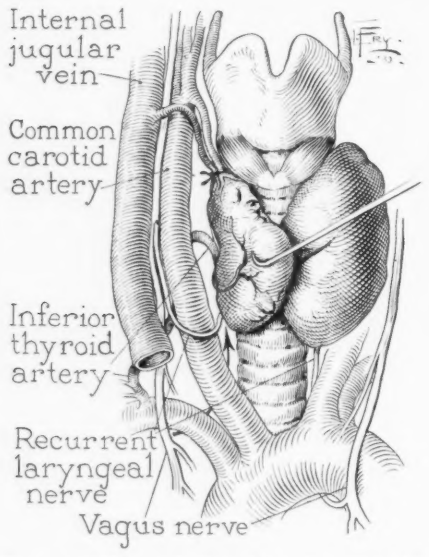

Fig. 1. Artist's reproduction of the anomalous origin of the right recurrent laryngeal nerve as found at operation. The right subclavian artery arising directly from the arch of the aorta is shown as a suspected concomitant anomaly.

Because of the rarity of such an anomalous origin of the right recurrent laryngeal nerve, we were interested in learning if it was described in the standard textbooks of anatomy or had been observed by others and reported in the literature. In Gray's *Anatomy* the origin of the nerve is described as follows: "The recurrent nerve on the right side arises in front of the subclavian artery; winds from before backward around that vessel, and ascends obliquely to the side of the trachea behind the common carotid artery, and either in front of or behind the inferior thyroid artery." Essentially the same description is found in Cunningham's, Davis', and Piersol's textbooks of anatomy. The only one which suggests the possibility of any variation in the origin of the nerve is Piersol's *Anatomy:* "In cases in which the subclavian artery arises dorsally, the right recurrent laryngeal passes directly downward and inward from the vagus to the larynx."

An article by Hooper, in 1887, described the anatomy and physiology of the recurrent laryngeal nerves in detail. He quoted a paper by Sted-

man, in 1823, in which was described the dissection of an aged female and the absence of the right recurrent laryngeal nerve. There were, however, nerves given off from the trunk of the vagus, about the middle of the neck, which passed directly to the larynx. There was also irregularity in the origin and course of the right subclavian artery. Hooper quoted Hart as reporting a case, in 1826, in which the subclavian artery arose directly from the aorta and the right recurrent laryngeal nerve came straight from the vagus and passed directly to the larynx instead of being recurrent. He quoted Hilton, Herard, Demarquay, Krause, Telgmann, and Brenner, as also having described similar variations in the right recurrent laryngeal nerve. In addition several of these authors described irregular origins of the subclavian artery.

Hooper stated that an irregular origin of the subclavian artery and of the right recurrent laryngeal nerve is common. He recorded the hypotheses advanced by early observers to account for this anomaly of the right recurrent nerve and discounted them all on the grounds that it can only be due to a developmental defect occurring during fetal life. He said: "The proof that the course of the recurrent nerves is a question of development is found in the fact that when, from any cause operating in early fetal life, irregularities of the arch of the aorta or in the origin of its primary branches exist, the recurrent nerves have always in such instances an anomalous origin and course."

The only report found in the more recent literature was that by Milianitch, in 1924, who described an anomalous arrangement of the great vessels arising from the arch of the aorta. He also described the right recurrent laryngeal nerve as arising from the vagus in the neck and passing almost horizontally to the thyroid gland. The left nerve was normal.

Other reports in recent literature, such as that of Fowler and Hanson, in 1929, Berlin and Lahey, in 1929, and Nordland, in 1930, have described the minute relationship of the recurrent laryngeal nerves to the inferior thyroid arteries, but the authors did not mention having observed an anomalous origin of the right recurrent nerve.

It would appear that an anomalous origin of the right recurrent laryngeal nerve is a relatively infrequent occurrence and has largely been lost sight of in recent literature, as is evidenced by the scarcity of such reports and by the fact that three of four standard textbooks on the subject of anatomy have failed to mention the possibility of such an anomaly.

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EDITORIALS

SURGERY, GYNECOLOGY AND OBSTETRICS

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MARCH, 1932

THE BIOLOGIC PREPARATION OF SURGICAL PATIENTS

PROBABLY the greatest progress in surgery during the last 25 years has been, not from improved operative technique or newer methods of antisepsis or asepsis, but from viewing the patient biologically.

In the beginning of this century, for instance, the mortality rate of operations for hyperthyroidism was high. The operation, technically considered, was more attractive than the procedures now used. The thyroid gland was exposed through a broad incision, the vessels and nerves were demonstrated much as in the dissecting room, ligatures and sutures were numerous and accurately placed, and the whole operation was a model of technical skill—yet many of these hyperthyroid patients died. The operation as done today is far cruder from a technical standpoint. No effort is made to expose the nerves or any particular vessel. A portion of the thyroid gland is left posteriorly without trying to demonstrate the recurrent laryngeal nerve as was formerly the custom. The transverse incision of the muscles of the neck has been generally abandoned, and no more sutures are placed than are necessary to hold the wound together—yet after the modern operation most patients recover.

The difference in this decreased mortality rate is not due to improvement in the technique, which has actually deteriorated from a mechanical and anatomical standpoint. It is due to the work of Plummer and others in finding that the toxic secretion from the hyperactive thyroid gland could be markedly altered by the administration of iodine. While the improvement may not be permanent, a change that often lasts for many weeks, occurs not only clinically but in the histology of the tumor. Operation with the patient under the influence of iodine is much safer than without iodine. It was found, too, that unnecessary trauma and exposure of tissue spaces are deleterious to the patient.

So in operations on the prostate gland. Removal of the prostate the day after the patient entered the hospital, which was not infrequently done 25 years ago, was followed by a high mortality rate. The operation was well executed but often the patient died. Gradually deflating the pressure caused by the obstructive prostate, draining the bladder until the kidneys could work efficiently against the new pressure conditions, decreasing the infection in the bladder by drainage, made the patients much safer for operation. Here, too, the technique of the operation has not improved.

It seems that we are approaching somewhat the same status in surgery of the large bowel. In anatomical and histological struc-

ture as well as in function, the large bowel is quite different from the small bowel, particularly the upper small bowel. With the right half of the colon acting as an absorbent of salts and water, and the left largely as a reservoir, there is practically no digestive action. This storage property also makes the colon an incubator. It teems with bacteria. If a resection is done without a preliminary enterostomy or preparatory treatment by diet or intraperitoneal injections, breaking down of the sutures and peritonitis are prone to occur. The blood supply of the colon is less than in the small intestine. This, too, must be taken into account when resection is indicated. After the mesentery of the loop to be resected has been divided and ligated, the mesentery should then be cut along its junction with the bowel until a spurting vessel occurs. This insures a good blood supply to the sutured colon. If complete enterostomy, after the old-fashioned method of bringing up the bowel on the abdominal wall and placing a glass rod under it, is done in the ascending colon 10 days or more before resection, the remaining portion of the colon receives absolute rest. The virulence and the number of bacteria are greatly diminished, the bowel contracts and bacteriologically the colonic contents are converted into the same condition as in the upper small intestine. Ten days or more after such a complete enterostomy, the resection can be done with an open technique as in the jejunum, and if the blood supply has been preserved, as mentioned, there need be no more fear of disaster than if the suturing were placed in the upper small intestine with its relatively sterile contents.

If the growth is large or the patient fat, the modified Mickulicz type of operation can be done, the mesentery being divided and ligated, the affected loop being brought up on the abdomen, the ends of the loop being clamped and excised with the cautery. After either type of operation, however, the physiologic rest provided by the complete colostomy should be continued for about 10 days after the union of the ends of the colon. The enterostomy, which should be done through a muscle-splitting incision, can be readily closed.

The application of biologic principles in excision of the colon seems to be just as desirable as in surgery of the thyroid or of the bladder and prostate. No matter how carefully the resection is done, by a closed method or otherwise, a bowel that still has the burden of function upon it and is loaded with fæces teeming with bacteria is not in the same favorable condition to resist infection or to heal as after a preliminary preparation which induces rest, removes the current of fæcal matter, and diminishes the bacterial contents to a minimum.

J. Shelton Horsley.

FASHIONS IN PROSTATECTOMY

As in many other conditions of varying pathology, there is no constant unanimity of opinion in regard to the best methods of relieving benign prostatic obstruction. At an earlier period perineal prostatectomy held the stage. Then the work of Magill, Belfield, Fuller, and Freyer pushed suprapubic prostatectomy into the limelight where it has occupied a larger portion of the stage than any other method. But for a good many years various methods of attack upon the types of benign obstructing prostate glands through the urethra have engaged the attention of experts. Beginning with the prostatic incisor of Bottini, continuing with the punch operation of Young, later modified into the cautery punch by Caulk, these transurethral methods have held out attractive possibilities. Considerable

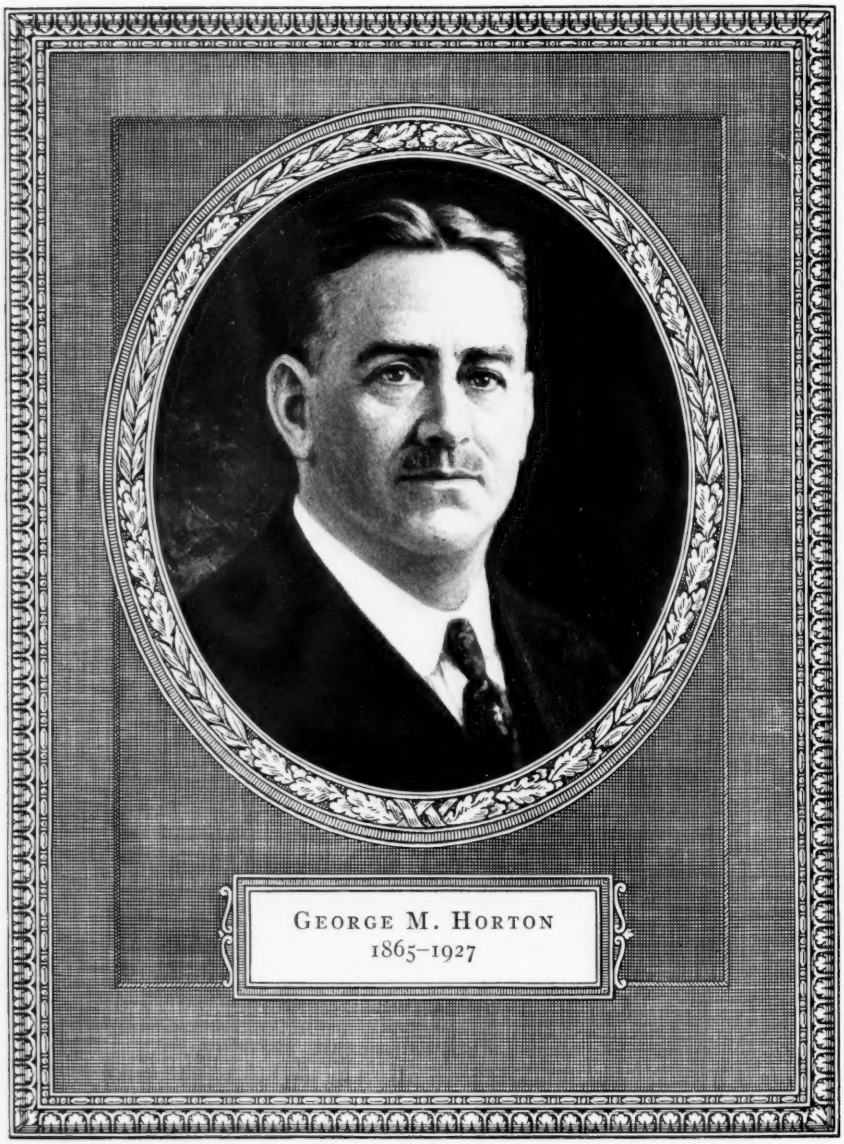
impetus has been given to them in the last few years by further modifications of instruments making possible better visualization of obstructing tissue and better control of hæmorrhage.

Unfortunately, the enthusiasm of experts and insufficient information on the part of the rank and file of the profession seem in some danger of pushing these transurethral methods of attack to the fore more rapidly than our knowledge and experience warrant. In the first place it is not always recognized that these procedures require rather unusual manual dexterity and they are almost certain to be occasionally undertaken by physicians whose training in urology, both diagnostic and operative, is insufficient. No operation on patients suffering from this disability will ever be free from risk. The precise risk in any individual case will depend partly on very complete knowledge of the condition of the patient not only as concerns his urinary tract but also as concerns his cardiovascular system and his resistance to the ravages of time, and partly on the skill and experience of the surgeon. There is a growing tendency to regard these operations as trivial since they can often be carried out by experts with a minimum of hospital confinement and risk. That they are not trivial and can never become so must be evident in view of the fact that the accidents of infection and bleeding will require all of the resources of the trained surgical urologist, and in view of the fact that these patients have a narrow margin of safety and may easily be pushed into eternity. There is grave danger that overenthusiasm in reporting immediate results of this procedure may place the more cautious surgeon in a very awkward position when he is called on to advise a patient who has been lightheartedly told that his difficulty can be cured by a trivial transurethral operation.

One does not have to be as old as Methuselah to remember the vicissitudes through which all operative attacks on the prostate gland have gone and the extent to which highly promising procedures have failed to live up to prediction. For the present the profession will be well advised to maintain a conservative attitude in regard to transurethral prostatectomy. It is perhaps true that it may be or may become the method of election in dealing with the relatively small though highly obstructive lesions, particularly those involving the glandular structures in the median line. That with improvements of technique the operation may prove satisfactory in a larger field is not improbable, but it would be a bold man who would assert that the present evidence would warrant this conclusion. If the method is cautiously employed by well-trained and dextrous surgeons it may achieve its position without the disappointments and catastrophes which have attended the enthusiastic receptions of most of the newer methods in this field.

For the present the older and better known methods, whether suprapubic or perineal, will, in the hands of most surgeons, prove better suited to the larger types of prostatic hyperplasia and will probably yield more lasting results. One may properly ask for this new candidate for favor in the field of operative technique a patient hearing, freedom from ill advised criticism, but also that it be not boldly pushed forward into a too prominent position from which retreat may be necessary and may prove embarrassing.

HUGH CABOT.



GEORGE M. HORTON
1865–1927

MASTER SURGEONS OF AMERICA

GEORGE M. HORTON

"HE was a born Doctor—a favored son of the gods of medicine," may well be the epitaph of Dr. George M. Horton, whose noteworthy medical career terminated on May 6, 1927, in Seattle, Washington, where he lived all but the first 5 of his 62 years. Dr. Horton was endowed by nature with great gifts, fitting him especially for his chosen profession to which he gave the best in him with a devotion seldom equalled.

Being a worthy servant of mercy, he made no distinction between the rich and the poor who daily thronged into his office. Undoubtedly he enjoyed one of the largest medical and surgical practices on the Pacific Coast. Hence, great is the multitude who mourn the passing of this skillful and much admired physician, who enjoyed the most valuable asset of a medical career—the affectionate regard of his patients.

Dr. Horton was born in Shabbona Grove, DeKalb County, Illinois, on March 17, 1865. His parents moved to Seattle when it was only a village of a few hundred inhabitants. The ancestral farm on which he spent his happy childhood is now named Georgetown in memory of this great pioneer son.

After receiving a preliminary education in the public schools of Seattle and at the Territorial University, and driven by the inborn urge to serve suffering humanity, he went to New York and studied medicine at the Bellevue Hospital Medical School from which he graduated in 1890.

At that time it was not obligatory to spend additional years in a hospital for practical training under the guidance of experienced masters. Hence, the youthful doctor returned immediately after graduation to his home town and took up the practice of medicine.

However, he had the good fortune to be associated with Dr. J. S. M. Smart, a physician of wide clinical experience who had been his medical preceptor before he entered the medical college. Unfortunately, this association terminated after a short time by the death of Dr. Smart and he was left alone to carry on as best as nature and a few years of study had equipped him. Through his innate fitness for the practice of medicine, good judgment, and indefatigability he soon won the confidence of the municipality and was made County Coroner for a term of 4 years.

He also devoted much time and thought to the fraternal organizations and in due time attained the thirty-second degree in Scottish Rite, the Knights Templar Degree in the York Rite, and became a member of Nile Temple of the Mystic Shrine, a member of Odd Fellows, United Workmen, Knights of Pythias and of Woodmen of the World.

Later also in the medical associations, he proved himself to be a just and an able leader, as president of the King County Medical Society, president of the Washington State Medical Association, and president of the North Pacific Surgical Association.

However, as his fame spread throughout the Northwest and the number of his patients increased, he felt forced to devote all his energies to the practice of medicine. Being a decided individualist he preferred to work alone. Therefore, his time was almost too full to allow much diversion, though once in a while he allowed himself the joy of witnessing a baseball game—the only sport in which he was keenly interested to the extent of sacrificing a few hours away from the office.

It was not uncommon for Dr. Horton to see 50 or 60 patients during an afternoon, in spite of the fact that it was his custom to lock the door of his reception room promptly at four-thirty in the afternoon.

Possessing an unusual sense of responsibility to his profession and to those who sought his services, he kept office hours also on Sundays in order to be available to anyone who could not consult him on week days. Unstinted service to his fellowmen was undoubtedly one of his outstanding characteristics. Though for a number of years he was known as a general practitioner, effectively covering all branches of medicine, it was as a surgeon that he became distinguished. He excelled particularly in abdominal surgery. He was a member of the Western Surgical Association, Pacific Coast Surgical Association, North Pacific Surgical Association, and a fellow of the American College of Surgeons.

There are many of his colleagues who would wish with me that this great physician and surgeon had been spared to serve humanity as few are able or willing to serve.

O. F. LAMSON.

THE SURGEON'S LIBRARY

REVIEWS OF NEW BOOKS

THE book on the *Diagnosis in Joint Disease*, by Allison and Ghormley,[1] is the first of its type to appear in English, and is, therefore, highly acceptable. It is in the form of an atlas which is supplemented by text. The authors offer the compilation of data from their experience in the study of 289 cases of arthritis over a period of 6 years. They present the clinical radiological, bacteriological, chemical, and pathological data in these cases. They approach the subject from the standpoint of three main issues, viz.: (1) the etiological factor, (2) the tissue of the joint primarily affected, and (3) the character of the tissue change. The authors emphasize the fact that one of the important necessities in the study of joint diseases is greater accuracy in methods of diagnosis, and they urge too a common basis of classification of diseased processes.

The subjects covered are discussed in the following order: the physiology and chemistry of joint structures; the classification of arthritis; the classification of joint diseases; tuberculosis; traumatic arthritis; arthritis associated with loose body formation; pyogenic infections of joints; arthritis of uncertain origin; proliferative arthritis; and degenerative arthritis. Considerable space is given to the subject of osteochondritis dessicans.

The reviewer regrets to state that the authors omitted the following conditions: syphilitic arthritis; tumors of the synovial membrane; tumors of bone and synovial outpouchings or evagination.

They classify arthritis into 2 groups: those of known and those of unknown origin. Those of known origin are divided into 4 classes, namely: (1) traumatic arthritis, (2) bacterial agent, (3) arthropathies, and (4) constitutional disturbances. Those of unknown origin are classified as: (1) proliferative arthritis, (2) degenerative arthritis, and (3) unclassified. They believe that any classification should be based on the etiological factors responsible and on the primary tissue changes which result. They emphasize that disease processes are not of the bone but in the bone. In all forms of arthritis, with only one exception—namely, the degenerative form—the disease process primarily affects, first, the synovial membrane which proliferates; and, second, the marrow and connective tissue which proliferates.

All tissue changes such as atrophy and hypertrophy, loose body formation, eburnation, and cartilage destruction are secondary changes which follow the process of synovial and marrow proliferation. The diagnosis of proliferative arthritis is positively made by tissue study and the finding of focal collections of lymphocytes in the marrow and synovial membrane.

Ghormley found that in 41 cases in which the diagnosis of tuberculosis of the joints was made, only 27, or 66 per cent, were proved to be tuberculous. In 52 cases in which the diagnosis of tuberculosis was set down as the probable cause of the joint disease, only 27, or 52 per cent, were proved to be tuberculous. In 42 cases in which tuberculosis was not considered as a possible cause, 23, or 55 per cent, were proved to be tuberculous. Twenty-nine per cent of all cases of arthritis remain uncertain of diagnosis after the completion of all clinical tests.

The book is of unusual size and is very attractively and handsomely produced. The illustrations include photographs, diagrams, roentgenograms, and numerous photomicrographs. The manuscript is well written and contains references to the best literature on the subject, including notably the works of Nicols and Richardson, Fisher and Ely.

There are several typographical errors, and some of the roentgenograms are upside down. Many of the roentgenograms do not reveal what the legends indicate, due, undoubtedly, to loss of detail in reproduction. The colored illustrations of gross pathological changes are not as good as the microscopic colored plates. Examination of the illustrations with a magnifying glass is helpful especially of the photomicrographs. It does not help very much in examining some of the roentgenograms.

This book can be recommended very highly. It should be of value to orthopedic surgeons, pathologists, and internists. PHILIP LEWIN.

THE *Text-Book of Neuro-Anatomy*,[2] by Professor Albert Kuntz, is designed to introduce beginning students to the subject. It will not be as useful to the advanced student or clinical man as the author's book on the autonomic nervous system. An attempt has been made to acquaint the reader with the simplicity of the fundamental plan of the vertebrate nervous system and to build upon this ground-work only what the author considers the essential details of neurology. Although most of the chapters are very brief and skeleton-like in lack of detail, those on the cerebrum and possibly the cerebellum are written rather more in full. In criticism of the text

[1] DIAGNOSIS IN JOINT DISEASE; A CLINICAL AND PATHOLOGICAL STUDY OF ARTHRITIS. By Nathaniel Allison, M.D., F.A.C.S., and Ralph K. Ghormley, M.D. New York: William Wood and Company, 1931.

[2] A TEXT-BOOK OF NEURO-ANATOMY. By Albert Kuntz, Ph.D., M.D. Philadelphia: Lea & Febiger, 1931.

it may be said that among the details that might be sacrificed are a few neurological conceptions, such as the mixed sensory and motor character of the dorsal vagus nucleus, which, if not entirely disproved, are at least open to serious doubt. Pure morphological descriptions might be relieved in places by the addition of more functional explanations.

On the whole the book is well planned and nicely prepared. Most of the illustrations, new ones as well as those from other authors, are excellent. Exception can only be taken to the photomicrographs of sections through the brain-stem which are somewhat too small, very dark, and lacking in even the major details. These are not as completely labelled as one might desire. WILLIAM F. WINDLE.

BOOKS RECEIVED

Books received are acknowledged in this department, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

LEHRBUCH DER ROENTGENDIAGNOSTIK. By H. R. Schinz, W. Baensch, and E. Friedl. Vols. i and ii. 3d rev. ed. Leipzig: Georg Thieme, 1932.

MANUAL FOR THE JEWISH DIABETIC. By William S. Collens, B.S., M.D. Foreword by Henry Joachim, M.D. New York: Bloch Publishing Company, 1931.

CONQUERING ARTHRITIS. By H. M. Margolis, M.D. New York: The Macmillan Company, 1931.

CHIRURGISCHE INDIKATIONEN FUER AERZTE UND STUDIERENDE. By Professor Dr. Karl Reschke. Part i. Berlin: F. C. W. Vogel, 1932.

ONE HOUR OF MEDICAL HISTORY. Compiled by Benjamin Spector, M.D. Boston: The Beacon Press, Inc., 1931.

TUMOURS OF THE BREAST; THEIR PATHOLOGY, SYMPTOMS, DIAGNOSIS, AND TREATMENT. By Sir G. Lenthal Cheatle, K.C.B., C.V.O., F.R.C.S., and Max Cutler, B.Sc., M.D. Philadelphia and Montreal: J. B. Lippincott Company, 1931.

A DOCTOR OF THE 1870'S AND 80'S. By William Allen Pusey. Springfield, Illinois and Baltimore, Maryland: Charles C. Thomas, 1932.

FUNDAMENTALS OF ORTHOPÆDIC SURGERY IN GENERAL MEDICINE AND SURGERY (HARVARD LECTURES). By Robert B. Osgood, M.D., F.A.C.S., and Nathaniel Allison, M.D., F.A.C.S. New York: The Macmillan Company, 1931.

ANNUAL REPORT OF THE SURGEON GENERAL OF THE PUBLIC HEALTH SERVICE OF THE UNITED STATES FOR THE FISCAL YEAR 1931. Washington, D. C.: United States Government Printing Office, 1931.

NOUVEAU TRAITÉ DE PATHOLOGIE CHIRURGICALE. Published under the direction of A. Basset, H. Costantini, G. Jeanneney, J. Maisonnet, G. Miginiac. Vol. ii, 1st and 2d part, Membres by J. Maisonnet. Paris: G. Doin & Cie, 1931.

HISTORY OF MEDICINE IN THE UNITED STATES. By Francis R. Packard, M.D. Vols. i and ii. New York: Paul B. Hoeber, Inc., 1931.

IMHOTEP TO HARVEY; BACKGROUNDS OF MEDICAL HISTORY. By C. N. B. Camac, M.D. Foreword by Henry Fairfield Osborn, Sc.D., LL.D. New York: Paul B. Hoeber Inc., 1931.

VARICOSE VEINS; WITH SPECIAL REFERENCE TO THE INJECTION TREATMENT. By H. O. McPheeters, M.D., F.A.C.S. 3d rev. and enl. ed. Philadelphia: F. A. Davis Company, 1931.

FEMALE SEX HORMONOLOGY; A REVIEW. By William P. Graves, A.B., M.D., F.A.C.S. Philadelphia and London: W. B. Saunders Company, 1931.

COURTS AND DOCTORS. By Lloyd Paul Stryker. New York: The Macmillan Company, 1932.

A RADIOLOGICAL STUDY OF THE PARA-NASAL SINUSES AND MASTOIDS. By Amédée Granger, K.C.B., K.C.I., M.D., F.A.C.R. Philadelphia: Lea & Febiger, 1932.

PRIVY COUNCIL MEDICAL RESEARCH COUNCIL. The Intervertebral Discs; Observations on their Normal and Morbid Anatomy in Relation to Certain Spinal Deformities. By Ormond A. Beadle. London: Published by His Majesty's Stationery Office, 1931.

THE PRINCIPAL NERVOUS PATHWAYS; NEUROLOGICAL CHARTS AND SCHEMAS WITH EXPLANATORY NOTES. By Andrew Theodore Rasmussen, Ph.D. New York: The Macmillan Company, 1932.

THE FUNDUS OF THE HUMAN EYE; AN ILLUSTRATED ATLAS FOR THE PHYSICIAN. By Ernest Clarke, C.V.O., M.D., F.R.C.S. London and New York: Oxford University Press, 1931.

A TEXT-BOOK OF MEDICINE FOR NURSES. By E. Noble Chamberlain, M.D., M.Sc., M.R.C.P. With a Foreword by Miss E. M. Musson, C.B.E., R.R.C. London and New York: Oxford University Press, 1931.

CLINICAL ROENTGEN PATHOLOGY OF THORACIC LESIONS. By William H. Meyer, M.D. Philadelphia: Lea & Febiger, 1932.

PROHIBITING MINDS; AND THE PRESENT SOCIAL AND ECONOMIC CRISIS. By Stewart Paton, M.D. New York: Paul B. Hoeber Inc., 1932.

THE STORY OF MEDICINE. By Victor Robinson, M.D. New York: Albert & Charles Boni, 1931.

SURGERY OF THE CHEST. By George F. Straub, M.D., F.A.C.S. Springfield, Illinois and Baltimore, Maryland: Charles C. Thomas, 1932.

FRACTURA INTER DELTO PECTORAL. By Dr. Alberto Baraldi. Rosario, Argentina: Talleres Graficos Pomponio 1931.

AMERICAN COLLEGE OF SURGEONS

THE WRITING OF SCIENTIFIC PAPERS IN THE PERIOD OF OPPORTUNITY

"THE practicing surgeon has little time for the writing of papers." The truth of this statement, made by a recent visitor to the College Library, is evidenced by the large number of men highly esteemed in their communities who have never found time to organize series of cases and make a careful study of interesting phases for the benefit of men throughout the medical world who are working on the same problems. However, the professional man who has material and records of value and who appreciates the advantage of a detailed and exact knowledge of the progress of others is ready to share his own experiences and results, and to set aside the time required for preparing and presenting the material in suitable form. Many who, until now, have found their days too crowded with the more pressing responsibilities of their professional life may during the present health "moratorium" (declared of necessity by at least a portion of the lay public) find time to undertake the task which has hitherto been postponed. In this period of opportunity, if we will but regard it as such, the services of the Department of Literary Research can be of assistance.

Diversity of requirements. The Department of Literary Research was planned to meet the varied needs of Fellows of the College and other members of the profession in the preparation of their scientific papers. Whatever the need of the writer may be, the Department is ready to supply it. If it is desirable to know the number of tumors reported, similar to the unusual one which has recently come to operation, and details of the history, pathological report, and classification in each case, the Department can make the necessary search. If the exact details concerning the origin of a new type of treatment are desired from the literature of 5 years ago, the Department will compile the bibliography and abstract the data. If a brief reference has been noted to experimental work which is described in detail elsewhere, the Research Department can help in locating it. If, further, the article is written in a foreign language, it can be translated by the Department. Whether it is necessary to know what was written two hundred years ago or what is being published in a foreign land today, this staff of experienced workers is prepared to obtain the information, thus lessening the burden of time and labor required to search for the material which, together with the doctor's daily experience, is the basis for the study of his present case and the background for his paper.

Personal service. It is the aim of the Department to serve each Fellow as completely and satisfactorily as though he were immediately available in an adjacent office for conference and advice. Accordingly each person sending in a request is asked to outline his requirements as definitely as possible. The *subject of research* is to be stated fully and the *type of work* desired, i.e., "bibliography alone," "bibliography and abstracts," or a "translation of a single article (reference enclosed)." In outlining a request for a *bibliography* it is important to indicate the number of years to be covered by the research, i.e., "two years," "ten years," or "back to the beginning of the use of this type of treatment."

If *abstracts* and *translations* are desired it is advisable to state which phases of the subject are of interest. For example, an article may contain a discussion of the history, etiology, pathology, symptomatology, diagnosis, differential diagnosis, treatment, prognosis, end-results, experimental work, or any combination of these factors. The one making the inquiry may be interested only in treatment and prognosis. Thus a complete translation of any one article might contain much extraneous material and but little of real interest to the inquirer. If the entire article is in point, a complete translation can be furnished; if not, a brief paragraph will inform the inquirer of the nature of the article and in a general way of its contents.

The Department is interested in knowing the *date at which the material is to be completed* so that the report will reach the inquirer in ample time for his needs. Here, again, it is wise to be specific. It is more satisfactory to the doctor and to the

Department to plan for the completion of the material before "March 20, 1932" than to work with the less definite date, "as soon as possible," in mind.

An *appropriation* for the work should be indicated also. The package library service (selected reprint material from the College files) is without charge to Fellows of the College. The work of compiling bibliographies, abstracting, and translating is done at cost, and hence, a definite appropriation for each piece of work should be made. The Department will then supply as much material as is possible for the amount specified (or less), and if additional data is desired the limit of expenditure can be raised after the first report has been received. It should be borne in mind that not even the terms "comprehensive" or "limited service" will mean the same thing to all people at all times.

The College Library will supply blanks for the convenience of those desiring to outline research. However, a definite statement of the requirement of the doctor is the factor of importance. Whatever the need may be connected with the writing of a scientific paper, it is the purpose of the Department to render assistance—that all may share in the benefits to be derived from the experience and study of each.